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(And Thanksgiving just 6 months away)

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Cordially,

Cecil Birtcher

Cecil Birtcher, President
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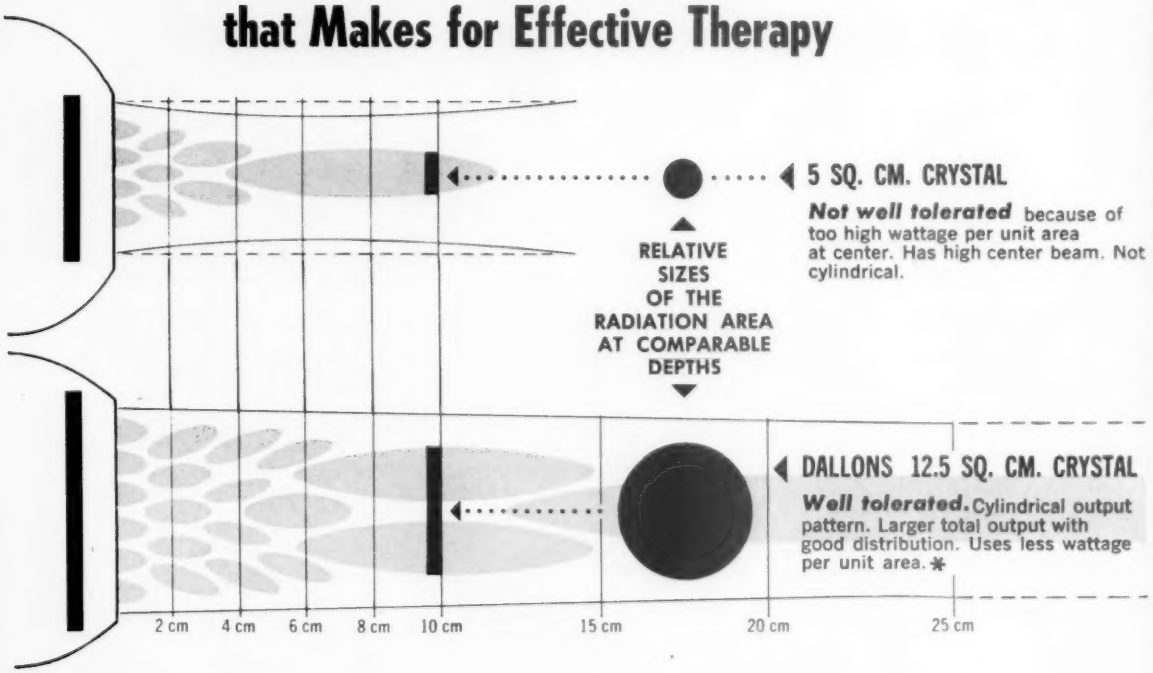

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Changes in Blood Flow, Oxygen Uptake and Tissue Temperatures Produced by the Topical Application of Wet Heat

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Chicago

● The effect of periods of 20 and 30 minutes of topically applied wet heat on blood flow, tissue temperature and oxygen uptake in the forearm was studied in 52 experiments performed on 51 normal male subjects. This modality produced a marked increase in local circulation, with all structures contributing to the response, including tissues located three or more centimeters below the surface of the skin. Associated with the augmentation in blood flow was a rise in skin, subcutaneous tissue and muscle temperatures. Short periods of exposure were not as effective as prolonged heating in elevating muscle temperature. During the application of wet heat for short periods of time there was a definite increase in oxygen uptake. It was pointed out that, even if the change had been of small magnitude, the use of wet heat in the presence of arterial insufficiency would still be contraindicated, mainly because of a loss of the cooling mechanism provided by the rapid increase in blood flow normally elicited by heat. As a result, there would be a trend toward an abnormally high rise in tissue temperatures, with a consequent elevation of metabolic needs which could not be satisfied. It is concluded that topically applied wet heat is a potent vasodilating agent in increasing local blood flow and in raising tissue temperatures even in deep structures. Moreover, it compares even more than favorably with some of the elaborate procedures commonly used in physical medicine for such purposes, provided high temperatures, up to 45 C., can be maintained.

Although topically applied wet heat has been utilized as a therapeutic agent for many centuries, there is still some question as to its action in this regard. One response on which there is agreement, however, is the marked increase in blood flow that follows even short periods of exposure.[1-5] On the other hand, a difference of opinion exists with respect to the degree of penetration that occurs with such a modality. A number of workers have found relatively slight heating of tissues below the surface of the skin,[6, 7] while others have reported changes at a much greater depth.[3, 5, 8-10]

The effect of heat on local oxygen uptake has been investigated by means of *in vitro* studies,[11, 12] and it has been reported that this agent raises the oxygen consumption of muscle slices. Similarly, prolonged exposure of the human forearm to wet heat causes approximately a twofold increase in oxygen uptake.[5]

The purpose of this study is to present the effects of short periods of topically applied wet heat on blood flow, tissue temperatures and oxygen uptake in the forearm. No attempt has been made to determine why such a modality is useful as a therapeutic tool or to implicate the physiologic responses investigated in the elucidation of this problem.

Method

The changes produced by the local application of 20 or 30 minutes of wet heat to the forearm were studied in 52 experiments, performed on 51 normal male subjects between the ages of 21 and 40 years. All external conditions and technics utilized in the determination of the various measurements were similar to those described in previous investigations.[5, 13] The duration of the test period averaged four hours, during which time no food was given to the subject.

In all but five experiments one or two veins in the antecubital space were cannulated by passing a thin-walled number 18 needle into each in the direction of the hand and threading a polyethylene catheter through it, so that the tip lay in the mass of tissue subsequently enclosed by the plethysmograph. The method of obtaining venous blood samples was similar to that previously described.[5] In 10 experiments only a

This investigation was supported in part by Research Grant H-2568 from the National Institutes of Health, United States Public Health Service.

From the Department of Physical Medicine and Rehabilitation, and the Department of Medicine, University of Illinois, College of Medicine, Chicago.

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superficial vein was cannulated; in 13, two superficial vessels were used; in 14, one or two deep veins were entered; while in 10, the procedure was carried out in both a superficial and deep vessel.

It is necessary to point out that, although there was generally very little difficulty identifying a vein draining surface tissues, this did not always apply to a deep vessel. Of importance in differentiating the two types of veins was the finding that blood from the one draining deep tissues almost always had a smaller oxygen content than that from superficial structures,[14] information, however, which was available only after cannulation had been carried out. Another response which was useful in this regard was the rate of change in oxygen content of the venous blood on exposure of the forearm to wet heat. There was an almost immediate increase in this figure in blood draining from superficial tissues while venous blood originating in deeper tissues showed a similar change only after the forearm had been exposed to heating for 10 or 15 minutes. The reason for the emphasis on the determination of the source of blood lies in its influence upon the derived values for oxygen arteriovenous difference, which will be discussed later.

Tissue temperatures were obtained by anchoring the skin, subcutaneous tissue and muscle thermocouples into their respective sites in the upper portion of the forearm,* subsequently enclosed by the plethysmograph, and inserting a modified thermocouple into the rectum for approximately eight centimeters.

Blood flow readings were recorded using a segment type of venous occlusion plethysmograph. The forearm was passed into the apparatus, so that the hand extended beyond it, the two openings were made water-tight according to the procedure previously described,[13] and the plethysmograph was filled with water at a temperature of 34 C. (bath temperature).

The plan of the experiment consisted of allowing the forearm to be exposed to the control bath temperature for 30 minutes, in order for equilibration to take place, and then collecting at least three sets of control readings of blood flow, tissue temperatures and venous oxygen content. (Arterial blood samples were not obtained, but instead venous blood was reoxygenated and the oxygen capacity determined. From this figure, arterial oxygen content was calculated[15] on the basis that arterial blood is 98 per cent saturated with oxygen.[16]) Following this the water was drained from the plethysmograph and immediately replaced with water at a temperature of 45 C., in order to produce heating of the segment of forearm in the apparatus. This higher level of temperature was maintained for a period of either 20 or 30 minutes, during which time the various measurements were repeated at intervals of three to four minutes. At the end of the period of exposure to heat the plethysmograph was drained and rapidly filled with water at the control level of 34 C. The various readings again were obtained at short intervals in the post-treatment period until they fell to or approached the control level. This took place approximately 65 minutes after therapy was terminated for both durations of heating.

Results

Symptoms and Systemic Responses. Application of wet heat to the forearm for 20 or 30 minutes usually produced no symptoms in the subjects; there was, however, occasional generalized sweating during the later portion of the treatment period. No consistent changes in blood pressure or pulse rate were noted either during or after the use of the modality; nor was there any significant alteration in rectal temperature.

Method of Determining Magnitude of Resultant Changes. As in previous studies,[5, 17-19] oxygen uptake for the periods before, during and after application of wet heat was calculated on the basis of blood flow and derived oxygen arteriovenous difference, using the Fick principle. The latter figure was the difference between the calculated reading for oxygen content of

*The muscle thermocouple tip was inserted into the brachioradialis muscle mass for an average distance of 3.4 centimeters (range of 2.4 cm. to 4.0 cm.) below the skin.

arterial blood and the measured oxygen content of the venous samples collected during the course of the experiment. In each experiment, graphs were constructed for the changes observed in oxygen uptake, as well as in the readings for blood flow and oxygen arteriovenous difference (figs. 1, 2).

In the case of blood flow and oxygen uptake, the areas under the positive deflections of the curve and above the baseline and those below the baseline and above the negative deflections were determined separately for the period of application of heat and for the post-treatment period, according to the method previously outlined.[17-19] In each instance the resultant change was recorded either as a positive or a negative quantity.

Control Levels. The average control data on blood flow, oxygen uptake and tissue temperatures are included in tables 1 to 3. The average resting oxygen content of venous blood draining superficial tissues was greater than that of blood from deeper structures (13.29 cc./100 cc. blood, as compared with 8.32 cc.), while the reverse was true for the readings of resting oxygen arteriovenous difference (0.0592 cc./cc. blood, as compared with 0.1019 cc.) and resting oxygen uptake (0.117 cc./min./100 cc. limb volume, as compared with 0.176 cc.).

Changes in Blood Flow. In the 41 subjects in whom the forearm was exposed to a bath temperature of 45 C. for 30 minutes, wet heat evoked an immediate increase in blood flow (fig. 1, table 1), causing the curve to rise sharply to a mean peak increase of 4.9 cc. per minute per 100 cc. limb volume, in excess of the average baseline of 2.0 cc. In almost all instances this maximal response occurred either in the last third of the period of application of heat or shortly after the procedure was terminated, the average time being 29

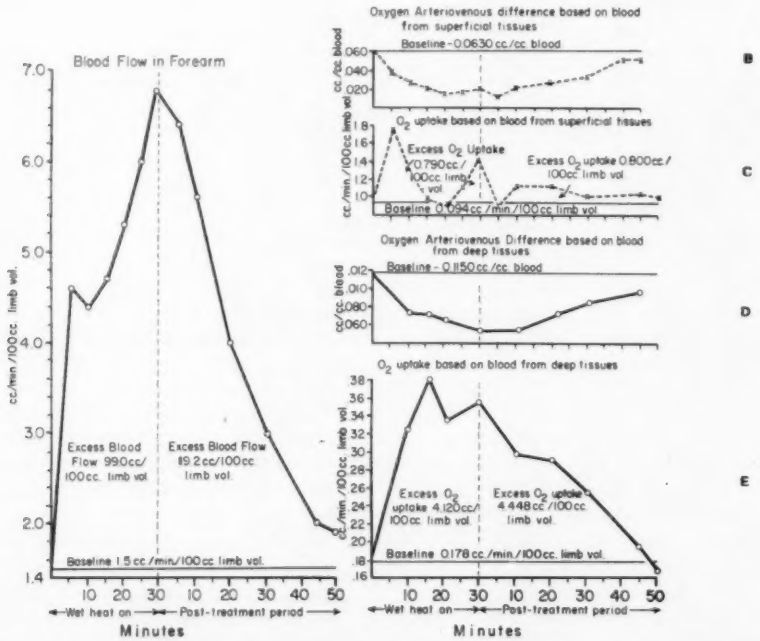

Fig. 1 — Effect of 30 minutes of topically applied wet heat to forearm of subject. A. Curve of blood flow changes. B. Curve of oxygen arteriovenous difference based on oxygen content of venous blood draining superficial structures. C. Curve of oxygen uptake based on oxygen content of venous blood draining superficial structures. D. Curve of oxygen arteriovenous difference based on oxygen content of venous blood draining deep structures. E. Curve of oxygen uptake based on oxygen content of venous blood draining deep structures.

minutes after initiation of the therapy. With the exception of 11 cases, in which the peak was reached two to 11 minutes after removal of heat, exposure of the forearm to a bath temperature of 34 C. produced a rather rapid and then, a somewhat slower, fall in the blood flow curve (fig. 1). For the group as a whole, the control level was reached on an average of 54 minutes after termination of heating.

During the treatment period, the average total blood flow augmentation, in excess of the control level, was 109.2 cc. per 100 cc. limb volume, while for the post-treatment period it was 130.6 cc. In 29 instances the magnitude of the increase in blood flow was greater in the post-treatment period than during the application of heat, while in the remaining 12 cases the reverse was true. For the group as a whole, the total excess blood flow elicited by the heat averaged 239.8 cc. per 100 cc. limb volume (table 1).

In the 11 subjects in whom the forearm was exposed to wet heat for a period of 20 minutes, the type of resulting blood flow change was grossly similar to that observed with 30 minutes. There was a sharp rise in the curve from an average baseline of 1.9 cc. per minute per 100 cc. limb volume to an average peak increase, in excess of the control level, of 4.4 cc. (fig. 2, table 1). This maximal response occurred on an average of 18 minutes after the bath temperature was raised to 45 C. In seven of the cases, exposure to the lower bath temperature produced an almost immediate drop in blood flow (fig. 2), while in the remaining four instances the readings continued to rise for several minutes and then fell. For the group as a whole, the baseline was reached on an average of 65 minutes after termination of heating.

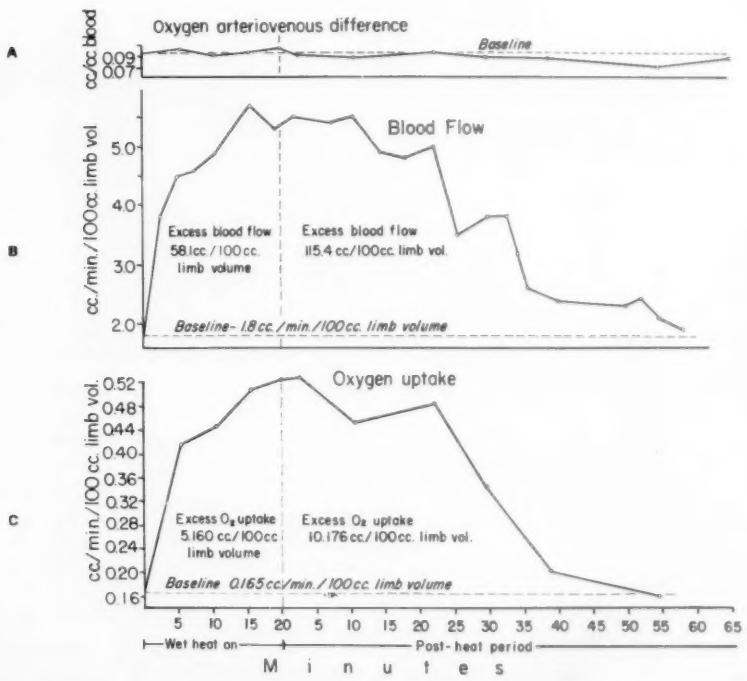

Fig. 2 — Effect of 20 minutes of topically applied wet heat to forearm of subject. A. Curve of oxygen arteriovenous difference based on oxygen content of venous blood draining deep structures. B. Curve of blood flow changes. C. Curve of oxygen uptake based on oxygen content of venous blood draining deep structures.

Table 1: Effect of Various Modalities on Blood Flow

	Wet Heat 20 min.	Wet Heat 30 min.	Wet Heat 120+ min.[5]	Mecholyl by Ion Transfer 30 min.[15]	Short-Wave Diathermy 30 min.[17]	Ultrasound 20 min.[19]
Control Level	1.9	2.0	2.6	2.1	1.7	2.1
Peak Increase	4.4	4.9	5.0	3.9	3.1	2.2
Time of Peak Increase	18	29	—	25	26	15
Total Increase During Therapy ..	51.2	109.2	—	92.8	36.4	20.5
Total Increase After Therapy ...	123.1	130.6	—	90.2	46.3	27.3
Sum of Increases	174.3	239.8	—	183.0	82.7	47.8
Period of Increase	85	84	—	83	66	46

During the period of application of wet heat for 20 minutes, the average total blood flow augmentation, in excess of the control level, was 51.2 cc. per 100 cc. limb volume, while for the post-treatment period it was 123.1 cc. In every instance but one the magnitude of the increase in blood flow was greater in the post-treatment period than during application of heat; in the one exception the reverse was true. For the group as a whole, the total excess blood flow elicited by the heat averaged 174.3 cc. per 100 cc. limb volume (table 1).

Changes in Oxygen Arteriovenous Difference. Results based on oxygen content of superficial venous blood: In the case of the 33 experiments in which oxygen arteriovenous differences were calculated on the basis of oxygen content of venous blood draining superficial structures, invariably exposure of the forearm to wet heat for 30 minutes resulted in a reduction in the magnitude of the readings (fig. 1). The average maximum fall from a mean control level of 0.0596 cc. per cc. of blood was 0.0143 cc. (a drop of 0.0453 cc.). This change was noted on an average of 31 minutes after initiation of the period of heating. In 15 of the cases the greatest fall occurred from four to 23 minutes in the post-therapy period. In the remainder this change was recorded within the last third of the treatment period. In each instance, after the maximum drop was noted, there was a gradual increase in the oxygen arteriovenous difference, with the baseline being reached or approached on an average of 52 minutes after termination of heating. In 15 of the 33 cases a positive deflection also was observed, with the peak increase occurring on an average of 46 minutes after termination of therapy. Following this change there was either a drop in the direction of the baseline or the curve remained at a point above the control level. The average total duration of the alterations in oxygen arteriovenous difference was 91 minutes after initiation of heating.

Results based on oxygen content of deep venous blood: In the case of the 14 experiments in which oxygen arteriovenous difference was calculated on the basis of oxygen content of venous blood draining deeper structures, invariably exposure of the forearm to wet heat for 30 minutes produced a decrease in the magnitude of the readings (fig. 1), although there was a delay in the first appearance of the alteration, as compared with the figures derived from oxygen content of superficial venous blood. The readings fell from an average control level of 0.1022 cc. per cc. of blood to 0.0626 cc. (a drop of 0.0396 cc.). Similarly, this maximal change occurred later than that observed in the case of the figures calculated on the basis of oxygen content of superficial venous blood (an average of 39 minutes after initiation of therapy, as compared with 31 minutes). In six instances it was noted just before or at the time of termination of exposure, while in the remaining eight it took place four to 47 minutes in the post-therapy period. In every experiment, after the maximal fall, there was a gradual rise in the curve

until the baseline was reached or approached on an average of 55 minutes after termination of treatment. In five of the 14 cases a positive deflection also occurred, with the peak increase being observed on an average of 36 minutes in the post-treatment period. Following this change there was a drop in the direction of the baseline. The average total duration of alterations in oxygen arteriovenous difference was 92 minutes after initiation of treatment.

The changes observed in the oxygen arteriovenous difference for 20 minutes of wet heat, based on oxygen content of deep venous blood, were in most instances grossly similar to those resulting from the longer period of therapy. During exposure to the modality, in eight instances all the readings became less than the control level, while in the remaining three they fluctuated on either side of the baseline (fig. 2). After termination of treatment, in nine cases the figures continued to be smaller than the control reading for varying periods of time and then they either returned to or approached the baseline, while in the remaining two instances the curve fluctuated on either side of it.

Changes in Oxygen Uptake. Results based on oxygen content of superficial venous blood: In 33 experiments on 32 subjects, the effect of 30 minutes of wet heat on oxygen uptake was studied, in each instance the latter figure being calculated on the basis of oxygen content of blood obtained from one or two veins draining superficial tissues (table 2). For the most part, no consistent pattern of change was noted, although the results could be placed into several categories depending upon the contour of the curves representing the changes. In eight experiments, during the application of heat all the oxygen uptake figures fell below the control level, to remain so for variable periods of time after termination of the treatment. Then they rose to reach the baseline, or, in some instances, to climb above it. In eight experiments the oxygen uptake curve during the period of heating consisted of several small peaks, in each case followed by a fall to or slightly above the control level (fig. 1C). In 10 experiments the oxygen uptake curve during the period of treatment fluctuated on either side of the baseline, in the form of small or large rises and falls. In five instances there was either a gradual or an abrupt increase in oxygen uptake during the period of heating, with a peak response being reached shortly before or at the end of the treatment. In the last case in the series the maximal effect, which was observed early in the treatment period, was followed by a drop in the direction of the baseline.

Table 2: Effect of Various Modalities on Oxygen Uptake

	Wet Heat 20 min.	Wet Heat 30 min.	Wet Heat 120+ min.[5]	Mecholyl by Ion Transfer 30 min.[15]	Short-Wave Diathermy 30 min.[17]	Ultrasound 20 min.[19]
Based on Venous Blood from Superficial Tissues						
Control Level	——	.117	.165	.141	——	——
Peak Increase	——	.080	.261	0.099	——	——
Time of Peak Increase	——	23	——	24	——	——
Total Increase	——	0.703	——	2.052	——	——
Period of Increase	——	92	——	82	——	——
Based on Venous Blood from Deep Tissues						
Control Level	.157	.176	.199	——	.118	.132
Peak Increase	.306	.354	.369	——	.109	.123
Time of Peak Increase	20	28	——	——	25	15
Total Increase	10.793	14.666	——	——	2.973	2.046
Period of Increase	76	94	——	——	58	43

For the group as a whole, the peak increase, in excess of the control level of 0.117 cc. per minute per 100 cc. limb volume, was 0.080 cc. This occurred on an average of 23 minutes after initiation of therapy (table 2). During the application of the wet heat, the average total oxygen uptake, in excess of the control level, was 0.245 cc. per 100 cc. limb volume, while for the post-treatment period it was 0.458 cc. In 14 cases there was an actual decrease rather than an increase in oxygen uptake during the period of application of the heat. In seven of these a similar type of change was observed in the post-therapy period, while in the remaining seven there was an increase in oxygen uptake during this time. In four other experiments an increase in excess oxygen uptake was noted during therapy but not afterward. In the remaining 14 experiments a rise in oxygen uptake occurred during both periods of observation. For the group as a whole, the total excess oxygen uptake elicited by the 30 minutes of heat was 0.703 cc. per 100 cc. limb volume (table 2).

It was considered of interest in the case of 13 subjects, to compare the changes produced by 30 minutes of exposure to heat, utilizing the oxygen content of blood samples from two different superficial veins in the same individual as a basis for the derivation of the oxygen uptake. In the case of the control level, the peak increase, and the total excess oxygen uptake during and after therapy, the readings obtained for the two sets of determinations were grossly similar, thus suggesting that different portions of superficial tissues appeared to respond in a similar manner to the stimulus of heat.

Results based on oxygen content of deep venous blood: In the case of the 14 experiments in which oxygen uptake was calculated on the basis of oxygen content of venous blood draining from deep structures, exposure of the forearm to wet heat for 30 minutes consistently produced an almost immediate and somewhat abrupt increase in the readings (fig. 1E), with an average peak rise of 0.354 cc. per minute per 100 cc. limb volume above the control level of 0.176 cc. (table 2). The maximum change took place on an average of 28 minutes after initiation of therapy. Except for five cases in which the peak effect was noted three to 18 minutes in the post-therapy period, exposure of the forearm to a bath temperature of 34 C. resulted in an immediate fall in the oxygen uptake curve (fig. 1E). For the group as a whole, the baseline was reached or approached on an average of 64 minutes after termination of therapy.

During the application of wet heat, the average total oxygen uptake, in excess of the control level, was 7.199 cc. per 100 cc. limb volume, while for the post-treatment period it was 7.467 cc. In nine of the cases the magnitude of the excess oxygen uptake was greater in the post-therapy period than during heating, while in five the reverse was true. For the entire group, the total excess oxygen uptake elicited by 30 minutes of heating was 14.666 cc. per 100 cc. limb volume (table 2). This is in contrast with 0.703 cc. per 100 cc. limb volume, the figure which represented the total excess oxygen uptake calculated on the basis of oxygen content of venous blood draining superficial tissues.

In the case of the 11 experiments in which wet heat was applied for 20 minutes, the oxygen uptake curves based on oxygen content of venous blood draining deep structures generally resembled those obtained with 30 minutes of exposure. The mean peak increase, which occurred on an average of 20 minutes after initiation of therapy, was 0.306 cc. per minute per 100 cc. limb volume, in excess of the control level of 0.157 cc. (fig. 2, table 2). After heating was terminated, the curve approached or reached the baseline on an average of 56 minutes after termination of heating. During application of the heat, the average total oxygen uptake, in excess of the control level, was 3.212 cc. per 100 cc. limb volume, while for the post-treatment period it was 7.581 cc., a figure which was similar to the one obtained with 30 minutes of heat for this period (7.467 cc.). For the group as a whole, the total excess oxygen uptake elicited by 20

minutes of heating was 10.793 cc. per 100 cc. limb volume (table 2).

Comparison of Oxygen Uptake Curves Based on Oxygen Content of Superficial and Deep Venous Blood. In 10 experiments both superficial and deep venous blood samples were collected before, during and after 30 minutes of exposure to wet heat. In each instance the oxygen uptake curves based on oxygen content of blood from the two different sources varied markedly. In the case of superficial venous blood, the average peak increase, in excess of the control level, was only 0.066 cc. per minute per 100 cc. limb volume, whereas a comparable figure based on blood from deep structures was 0.386 cc. Furthermore, the total excess oxygen uptake for the entire period of change based on superficial venous blood was 0.768 cc. per 100 cc. limb volume, as compared with 16.080 cc., the figure derived from oxygen content of deep venous blood.

Changes in Tissue Temperature. Skin Temperature: In the 40 subjects in whom the effect of 30 minutes of application of local wet heat on tissue temperature was studied, the skin temperature increased on an average of 6.4 C. from a control level of 34.7 C., with the peak response occurring on an average of 19 minutes after initiation of treatment (table 3, fig. 3). In those instances in which this point was reached before termination of therapy, the readings then remained at the level of the maximum effect until the forearm was exposed to the control temperature of 34 degrees. When heat was removed, there was at first a rapid and then a more gradual fall in skin temperature (fig. 3), with the baseline being reached on an average of 48 minutes after termination of the treatment.

In the 10 experiments in which the effect of 20 minutes of application of local wet heat was studied, the changes in skin temperature were grossly similar to those observed with 30 minutes of exposure. The readings increased on an average of 6.7 C. from a control level of 34.6 C., with a peak response occurring on an average of 13 minutes after initiation of the treatment (table 3, fig. 4). As in the case of the longer period of exposure, the curve remained at the level of the peak response until the forearm was exposed to the lower bath temperature. After the latter step was carried out, the readings began to drop, rather sharply at first and then more

Table 3: Effect of Various Modalities on Tissue Temperature

Site	Wet Heat 20 min.	Wet Heat 30 min.	Wet Heat 120+ min.[5]	Mecholyl by Ion Transfer 30 min.[15]	Short-Wave Diathermy 30 min.[17]	Ultrasound 20 min.[19]
Skin, Degrees Centigrade						
Control Level	34.6	34.7	33.4	34.4	34.6	34.9
Peak Increase	6.7	6.4	8.3	0.5	1.3	0.9
Time, Peak Increase	13	19	——	28	24	16
Duration of Increase	84	78	——	83+	64	48
Subcutaneous Tissue, Degrees Centigrade						
Control Level	34.9	34.6	34.0	34.9	34.7	35.1
Peak Increase	5.1	5.4	6.4	0.6	1.5	1.4
Time, Peak Increase	15	23	——	27	26	14
Duration of Increase	79	86	——	83+	69	44
Muscle, Degrees Centigrade						
Control Level	36.1	35.8	35.8	35.6	36.2	36.3
Peak Increase	1.4	1.8	2.8	0.4	1.9	0.9
Time, Peak Increase	21	29	——	33	27	17
Duration of Increase	77	89	——	83+	72	57

gradually (fig. 4), until the baseline was reached at an average of 64 minutes after termination of the treatment.

Subcutaneous tissue temperature: During the application of wet heat for 30 minutes, the subcutaneous tissue temperature increased on an average of 5.4 C. from an average control level of 34.6 C., with the maximal effect being noted on an average of 23 minutes after initiation of the treatment (table 3).

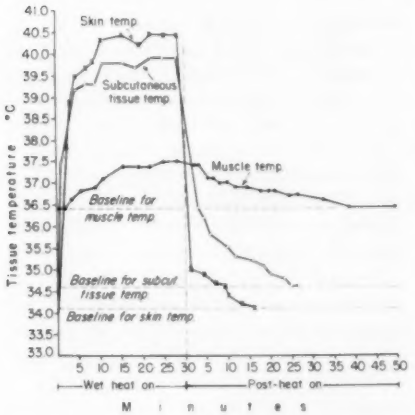

Fig. 3 — Curves representing changes in skin, subcutaneous tissue and muscle temperatures, obtained during and after 30 minutes of topically applied wet heat to the forearm.

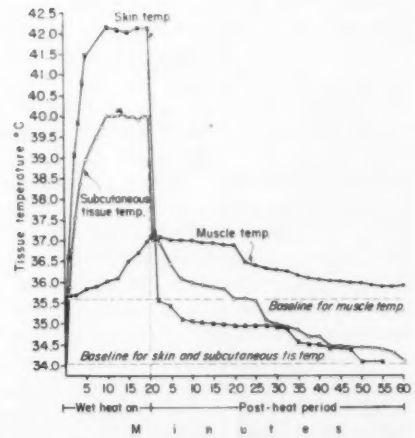

Fig. 4 — Curves representing changes in skin, subcutaneous tissue and muscle temperatures, obtained during and after 20 minutes of topically applied wet heat to the forearm.

The readings then generally remained at the level of the peak rise until the heat was removed. At that time they immediately began to fall toward the control level, reaching it on an average of 56 minutes after termination of the treatment.

The application of wet heat for 20 minutes produced approximately the same type of response in subcutaneous tissue readings as did the longer period of exposure. An average peak increase of 5.1 C., from an average control level of 34.9 C., was reached on an average of 15 minutes after initiation of the treatment (table 3). Again the readings remained at the level of the peak rise until the heat was removed (fig. 4). At that time they began to fall gradually toward the control level, reaching it on an average of 59 minutes after termination of the treatment.

Muscle temperature: With 30 minutes of application of heat, the muscle temperature readings rose more slowly than did the other two values (fig. 3), to attain a mean peak increase of 1.8 C. from an average control level of 35.8 C. (table 3) on an average of 29 minutes after initiation of heating. In contrast to the readings for skin and subcutaneous tissue temperatures, in six instances the peak response occurred after therapy had been terminated. On exposure of the forearm to the lower bath temperature, there was a gradual drop in the muscle temperature, with the baseline being reached on an average of 59 minutes after termination of therapy.

With 20 minutes of application of heat, muscle temperature readings also rose slowly (fig. 4), to attain a mean peak increase of 1.4 C. from a control level of 36.1 C.; the maximum effect was noted on an average of 21 minutes after initiation of heating (table 3). The substitution of water at 34 C. produced a fall in the readings, with the baseline being reached on an average of 57 minutes after termination of treatment.

Discussion

Results of the present study support the generally accepted view that topical

wet heat is a potent vasodilating agent. The application of this modality for 20 or 30 minutes produced more than a twofold augmentation in blood flow to the forearm, with an increased circulation persisting for approximately one hour after termination of the period of exposure. The average total excess blood flow elicited by 20 minutes of heating was equivalent to the quantity which would normally have entered the forearm during a period of approximately 92 minutes (174.3 cc., the average excess blood flow per 100 cc. limb volume, divided by 1.9 cc., the average control blood flow per minute per 100 cc. limb volume). For 30 minutes of heating the comparable figure was 120 minutes (239.8 cc., the average excess blood flow per 100 cc. limb volume, divided by 2.0 cc., the average control blood flow).

Comparison of the data on blood flow in the present study with those obtained in a previous investigation, in which wet heat was applied to the forearm for about two hours,[5] reveals that prolonged exposure had very little effect on increasing the peak response beyond the levels attained with the two shorter periods of therapy (table 1). These findings are in accord with those of Barcroft and Edholm,[3] who reported that at 45 C. the blood flow rose to a maximum within 30 minutes and then either remained almost constantly at this level or increased only slightly over the next two hours of heating. The present data also point out that, in the case of the duration and magnitude of increase in blood flow in the post-therapy period, there was very little difference between the response to 20 and 30 minutes of heating. However, a greater total augmentation in local circulation was noted with 30 minutes, as the result of a more marked change during the treatment period. Nevertheless, when the results were calculated on the basis of one minute of exposure, the unit increase in blood flow for the shorter period of therapy was somewhat greater than that for the longer one (8.7 cc. per 100 cc. limb volume, as compared with 8.0 cc.).

It can be stated, therefore, that prolonged application of wet heat produces neither a proportionate rise nor a fall-off in blood flow and that, aside from the fact that the duration of the enhanced circulation is longer, there is no particular advantage to its use over shorter periods of exposure. In fact, when all factors are considered, heating for 20 minutes would appear to be an almost optimal period of time for clinical application, if an adequate increase in blood flow is desired.

It is of interest to compare the average total excess blood flow response to topical wet heat (239.8 and 174.3 cc. per 100 cc. limb volume for 30 and 20 minutes of exposure, respectively) with the changes observed in the case of other physical modalities studied in the same manner (table 1). In previous investigations it was found that the total excess blood flow elicited by 30 minutes of short-wave diathermy was an average of 82.7 cc. per 100 cc. limb volume,[17] while for Mecholyl by ion transfer, applied for the same period of time, it was 183.0 cc.[18] Exposure to ultrasound for 20 minutes resulted in a total blood flow increase of 47.8 cc.[19]

The finding that in most instances the oxygen arteriovenous difference became smaller with exposure to heat can be explained on the basis of a more rapid volume rate of blood flow through the tissues, resulting in less oxygen being removed from each unit volume of blood. The fact, therefore, that, aside from the delay in the appearance of the change, the values calculated from the oxygen content of venous blood draining from deep tissues were affected in a manner similar to those based on oxygen content of blood from superficial structures supports the view that the vascular tree in the muscles also contributed to the increase in circulation produced by topical wet heat. The possibility that alterations in oxygen uptake resulting from heating of the tissues could also have materially altered the oxygen arteriovenous difference is not supported by the evidence, since under such circumstances there would have been an increase rather than the reported decrease in this measurement.

It can therefore be inferred that the predominant influence upon oxygen arteriovenous difference during and after heating was the augmentation in blood flow through both superficial and deep tissues. Furthermore, any resulting elevation in oxygen uptake of the tissues produced by the heat was probably satisfied primarily by this increase in local circulation, with the mechanism of greater removal of oxygen from each cc. of blood playing little or no role.

The fact that in the present study the oxygen uptake changes produced by wet heat varied markedly, depending upon whether the figures were based on the oxygen content of venous blood draining superficial or deep tissues (table 2), requires an explanation. In this respect it is of interest to point out that if the metabolic requirements of tissues remain constant, an increase in local blood flow theoretically could result in a proportionate decrease in oxygen arteriovenous difference, so that the derived value for the oxygen uptake figure would be unchanged. Such a possibility could apply in the present study to the situation in which minor calculated increases in oxygen uptake, based on oxygen content of superficial venous blood, were associated with marked augmentations in blood flow. However, it must be pointed out that the increased cutaneous circulation was not reflected proportionately in the total blood flow figure, since the latter was the resultant of vascular changes not only in the superficial tissues, but also in the muscles, in which the vasodilating effect of the surface heat was not as great, at least, initially. It would therefore be expected that the oxygen uptake figure would be smaller than the true reading when derived from an oxygen arteriovenous difference primarily influenced by vascular changes in superficial tissues, and hence that it would be too low to be representative of the total alteration.

With regard to the calculations of oxygen uptake based on blood draining from deep tissues, the situation was reversed. Under these circumstances a much higher than representative oxygen arteriovenous difference was obtained, due to the less marked vascular response elicited in the muscles by topical heat. At the same time, the figure for total blood flow did not proportionately reflect the vascular changes produced in the deep tissues by the heat, because of the contribution of the greatly enhanced cutaneous circulation. Hence, an oxygen uptake figure derived from such data would most likely be greater than the true value.

It would seem, therefore, that the reported excess increase in oxygen uptake based on blood from superficial tissues was probably too low, while that based on blood from deep tissues was probably too high. In view of the fact that with the short periods of heating, the effect of this modality on muscle was much less than that on superficial tissues, it is quite possible that the true uptake figure, representative of the over-all response of the forearm to this modality, was closer to the reading based on blood draining skin and subcutaneous tissue than that derived from blood originating in muscle.

Ideally, an accurate oxygen uptake figure during heating could have been determined if the readings for oxygen arteriovenous difference were based on a mixed sample of venous blood, made up of allocates from all structures responsible for the increase in blood flow, in proper proportion to their contribution to the total change. However, since such an aim could not be achieved in the human subject, the only other alternative was the one utilized in the present study.

Although there is basis for a comparison of blood flow changes produced by various types of modalities, a similar study of the resulting alterations in oxygen uptake gives much less pertinent information, mainly because of the marked variations observed in this figure with wet heat, depending upon whether the venous blood used in the derivation of the oxygen arteriovenous difference drained from superficial or deep structures. Nevertheless, it is of interest to point out that the excess oxygen uptake produced by 30 minutes of short-wave diathermy, based on oxygen content of blood from deep tissues, was 2.973 cc. per 100 cc. limb volume[17] (table 2), and

that for Mecholyl by ion transfer, based on oxygen content of blood from superficial tissues, was 2.052 cc.[18] In each set of experiments the structures drained by the blood utilized in the calculations are considered to represent the major locus of action of the modality. As already mentioned, in the case of 30 minutes of wet heat, the excess oxygen uptake figure derived from the oxygen content of superficial venous blood was 0.703 cc. per 100 cc. limb volume, while that based on oxygen content of deep venous blood was 14.666 cc.

It is necessary to point out that even if short periods of topically applied wet heat were to produce only small increases in oxygen uptake, their use would still be contraindicated in the presence of arterial insufficiency. Under such circumstances, the cooling effect normally provided by the greater blood flow elicited by heat is minimal, due to irreversible changes in the arterial tree, and hence the temperature of the exposed structures will tend to approach that of the surrounding water bath. Consequently, oxygen needs of the tissues will rise, with little or no means available to satisfy them, a situation which is conducive to death of poorly viable structures. Furthermore, aside from anoxia, heat may produce irreversible changes in the protein-colloidal system.

Comparison of the data on the changes in tissue temperature produced by 20 and 30 minutes of exposure with those obtained in a previous study, in which the forearm was subjected to wet heat at 45 C. for at least two hours,[5] reveals that prolonged heating had very little effect on increasing skin temperature beyond the level attained with the shorter periods (41.7 C., as compared with 41.3 C. and 41.1 C. for 20 and 30 minutes, respectively) (table 3). Similar results were noted in the rises in subcutaneous tissue temperature (40.4 C., as compared with 40.0 C. for both 20 and 30 minutes of exposure). Only in the case of muscle temperature was a higher level attained (38.6 C., as compared with 37.5 C. for 20 minutes and 37.6 C. for 30 minutes). Also of interest in this regard is the finding that for 20 and 30 minutes of heating, the maximal levels were approximately the same.

Such data therefore support the belief that topical heat can raise the temperature of deeper structures (at least to an average distance of 3.4 cm. beneath the skin) and that prolonging the period of application produces a higher muscle temperature. However, it cannot be anticipated that this level would rise much above the temperature of the blood, since the marked vasodilating effect on muscle vessels initiated by the heat would act as a mechanism for its removal via the blood stream.

It is of interest to compare the tissue temperature changes produced by topical application of wet heat for 30 minutes with those elicited by other modalities studied in a similar manner (table 3). The rises in skin and subcutaneous tissue were much higher with wet heat (6.4 C. for skin; 5.4 C. for subcutaneous tissue) than with either Mecholyl by ion transfer (0.5 C. for skin; 0.6 C. for subcutaneous tissue)[18] or with short-wave diathermy (1.3 C. for skin; 1.5 C. for subcutaneous tissue).[17] In the case of changes in muscle temperature, the increase with wet heat (1.8 C.) was only slightly less than that with short-wave diathermy (1.9 C.) and considerably more than that with Mecholyl by ion transfer (0.4 C.).

Summary and Conclusions

The effect of periods of 20 and 30 minutes of topically applied wet heat on blood flow, tissue temperatures and oxygen uptake in the forearm was studied in 52 experiments performed on 51 normal male subjects.

This modality produced a marked increase in local circulation, with all structures contributing to the response, including tissues located three or more centimeters below the surface of the skin. Prolonged heating appeared to have no particular advantage over shorter periods of application in eliciting an optimal vascular effect.

Associated with the augmentation in blood flow was a rise in skin, subcutaneous tissue and muscle temperatures. Short periods of exposure were not as

effective as prolonged heating in elevating muscle temperature.

During the application of wet heat, the magnitude of the average total increase in oxygen uptake derived from the oxygen content of blood draining deep tissues was found to be 14.666 cc. per 100 cc. limb volume, while that based on venous blood from superficial structures was 0.703 cc. It was believed that the true change in oxygen uptake, representative of the over-all response to wet heat, was closer to the reading based on blood draining skin and subcutaneous tissue than that derived from venous blood originating in muscles.

It was pointed out that even if short periods of locally applied wet heat were to produce only small increases in oxygen uptake, in the presence of arterial insufficiency, the use of the modality was still contraindicated. This was so because there was a loss of the cooling mechanism provided by the rapid increase in blood flow normally elicited by heat, and hence a trend toward an abnormally high rise in tissue temperatures, with a consequent elevation of metabolic needs which could not be satisfied.

It is concluded that topically applied wet heat is a potent agent in increasing local blood flow and in raising tissue temperatures, even in deep structures. Furthermore, it compares more than favorably with some of the much more elaborate procedures commonly used in physical medicine for such purposes, provided high temperatures, up to 45 C., can be maintained.

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Myotonia Atrophica: Electromyographic and Endocrine Studies

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● This paper describes a mother and her son and daughter with myotonia atrophica. The mother showed a late onset of her disease and a masking of atrophy by obesity and coincidental diabetes mellitus. The son and daughter showed low 17 hydroxysteroid excretion; the son excreted subnormal amounts of 17 ketosteroids in the urine. Therapy with Meticorten, thyroid and triiodothyronine produced no beneficial effect. The description of the patients and the disease include discussion of muscular involvement, endocrine changes, cataracts, and cerebral changes.

Myotonia atrophica (dystrophia myotonica) is an inherited disease manifested by muscular atrophy, myotonia, gonadal atrophy and cataracts. Inheritance is dominant and is said to display potentiation and anticipation in successive generations. A mother, son and daughter with this disease are described in this report.

Mrs. M. S., 64, was seen in 1957 because of muscular weakness of all four extremities since the age of 59. This progressed so that she was unable to walk without help, and was totally confined to a wheelchair. She could not get out of a chair without help, but stood up unaided. She was unable to walk up stairs and could hold only light objects in her hands. She had no difficulty in swallowing but had difficulty coughing up sputum. She had occasional numbness and tingling of her hands. Diabetes mellitus had been diagnosed 10 years ago and was controlled by diet and 44 units of NPH insulin for several years. She had gained 50 pounds in the past four years. Progressive loss of vision had occurred in the past two years. A goiter had been removed at the age of 24; following this she took one thyroid grain daily. Bilateral salpingoophorectomy and hysterectomy had been done at age 47. Right upper lobectomy had been performed for pulmonary tuberculosis at age 58 followed by antituberculous chemo-therapy.

Examination showed the patient to be obese. The face was expressionless with some ptosis of the lids and corners of the mouth. She had a nasal voice. Bilateral cataracts were present. A thyroidectomy scar was seen in the neck. The heart was not enlarged; no murmurs were heard. The heart rate was 60 per minute; the blood pressure was 130/90. A right thoracotomy scar was present. No atrophy of facial muscles was discernible. There was definite atrophy of the hands with a peculiar flabby feel to the muscles. The skin of the hands showed a translucency with prominence of the venules. There was profound weakness of all four extremities. Biceps and triceps reflexes were not obtained. Knee and ankle reflexes were diminished. A myotonic response was obtained on striking the gastrocnemius muscles and thenar eminences with a reflex hammer. Vibratory sense was absent over the left leg.

The patient was controlled by a 200 gram carbohydrate diet and NPH insulin of 50 units daily. She was put on Meticorten, 10 mg. three times each day, and felt that muscle strength was improved, but no objective evidence of the change could be seen.

Following discharge she continued a wheelchair existence, continued with thyroid and insulin but discontinued her Meticorten therapy. She fell and fractured her right ankle on Feb. 20, 1958. This healed after the application of a cast. She was rehospitalized on Dec. 23, 1958, because of an upper respiratory infection of one week's duration associated with difficulty in coughing up sputum, gagging and vomiting. She was discharged on Dec. 27, 1958. On Feb.

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3, 1959, she developed an increasing cough and dyspnea, expiring suddenly on Feb. 5, 1959.

Mrs. M. K., 33, the daughter of M. S., came to the office because of infertility. She had been married for six years. She did not consider herself to be ill. On questioning she admitted that she had never been able to move her furniture about. However, she had solved that problem by marrying a piano mover. She also had noted that she had difficulty in elevating her head when lying in bed. She had never been aware of any difficulty in releasing her grip.

Examination showed her to have the characteristic facies of myotonia atrophica with sunken temporal areas, drooping eyelids and corners of the mouth. She had a nasal voice. There was marked atrophy of the sternoclavicular head of the sternomastoid muscles. Striking atrophy of the forearms and legs was present. There was also atrophy of the hand muscles. Deep tendon reflexes were active. There were no sensory changes. There was a myotonic response of the gastrocnemius and thenar muscles on percussion with the reflex hammer. She was unable to suddenly extend her fingers after making a tight fist, the fingers moving as if immersed in tar. A loud systolic click was present over the apex of the heart. Blood pressure was 104/70. Pelvic organs were normal.

During a year's observation the patient has continued unchanged. She was put on Cytomel 25 micrograms daily but derived no subjective or objective benefit from it.

W. S., 32, the son of M. S., was asked to come to the office because of the history from his sister that he resembled her very closely. He considered himself in good health. He had been married for seven years and had two daughters. However, he had been separated from his wife and children for two years. On questioning, he admitted some weakness of his hands since the age of 17. He also had had a nasal voice since childhood. Examination showed a typical facies of myotonia atrophica with hollowed out temporal areas, drooping eyelids and mouth, thin oval face. He also had a nasal speech. There was marked atrophy of the clavicular heads of the sternomastoid muscles, atrophy of the forearm muscles especially the ulnar surface, atrophy of the thenar and hypothenar eminences, and atrophy of the legs. Tendon reflexes were normal. He had a myotonic response on mechanical stimulation of the calves and thenar eminences. He demonstrated the inability to rapidly extend his fingers after making a tight fist. Heart sounds were normal. Blood pressure was 96/54, the rate was 52/minute. His testes definitely were smaller than normal. The prostate was small. There was no recession of his hair line.

He was put on one thyroid grain daily for several months without any evident change in his condition.

Family Tree

No other members of the family were examined, but a history of other members of the family was obtained going back to the grandfathers and grandmothers of the mother, M. S., and her husband. No evidence of myotonia atrophica could be inferred from the history, though the families were large. There was a frequent occurrence of diabetes mellitus in both branches. There were five children in the family including patients M. K. and W. S. Two sons, 39 and 35 years old, and a daughter, 28, were normal judging from history and appearance. The two normal sons and daughter had a total of eight children who were all normal. M. K., the daughter, was childless.

The infrequency of occurrence of myotonia atrophica in this family tree is difficult to reconcile with the known dominance of inheritance. No doubt the condition may have existed without being recognized. Both M. K. and W. S. consider themselves well and do not recognize any muscular difficulty. The mother, M. S., did not have any difficulty until she was 59 years old.

Not only is inheritance considered to be dominant in this disease, but it is said to show anticipation and progression. Anticipation refers to the disease manifesting itself at an earlier age with each generation. It appears to be exemplified

in the mother manifesting the disease at age 59 and the children demonstrating the disease at ages 33 and 32. However, Thomasen[1] points out an alternative explanation, namely, the process of selection in the foregoing generation. Only the least affected patients are able to produce children. Therefore the parents will usually have a milder disease than the children. Progression, that is worsening of the disease with each generation, can be explained on the same basis as anticipation.

Muscular Involvement

The most characteristic feature of this disease is the muscle involvement. This consists of both myotonia and muscle atrophy. The myotonia appears in the form of an involuntary, protracted contraction of a striated muscle, caused either by voluntary, mechanical or electrical stimulation of the muscle and accompanied by a characteristic electrical phenomenon. Myotonia appears in two diseases, myotonia congenita or Thomsen's disease and myotonia atrophica. Thomsen's disease is restricted to muscles and is associated with muscular hypertrophy; it appears in childhood; it is generalized; and the myotonia grows less with age. Myotonia atrophica displays endocrine and ocular changes; it is associated with striking atrophy; myotonia is focal; the onset is at about 20 years of age. Myotonia lessens with the development of atrophy. In myotonia atrophica the myotonic response is seen chiefly in the finger flexors, tongue and gastrocnemius muscles. The myotonia may manifest itself by slowness of release of grasp, such as grasping a doorknob and not being able to let go for several seconds. It may be elicited by striking the calf, thenar eminence or tongue with a reflex hammer. Normally the rapid contraction and relaxation occurs within a second. The myotonic response consists of a rapid contraction which persists and slowly relaxes over a period of five to 30 seconds.

The muscular atrophy involves chiefly the face, sternomastoid and forearm muscles. The quadriceps and anterolateral leg muscles uncommonly show atrophy. A marked hollowing out of the temporal fossae occurs due to severe muscle atrophy. The levator palpebrae muscles atrophy with resultant ptosis. The orbicularis oculi muscles share in the atrophy causing sunken eyes. The masseter muscles atrophy, resulting in difficulty in chewing, a constantly open mouth and a forward thrust of the chin. Pharyngeal muscles atrophy with resultant nasal speech and dysphagia. There is striking atrophy of the sternoclavicular heads of the sternomastoid muscles, resulting in bobbing of the head backward when the patient arises from a horizontal position. Atrophy of the muscles of the forearm and hands occurs. Patients have difficulty in buttoning clothes as a result. Leg atrophy is much less common. The son and daughter in this group showed the changes of muscle atrophy strikingly and could be recognized as having myotonia atrophica at a glance. However, the mother failed to show these characteristic muscle changes. This was believed to be due to the masking effect of her obesity which filled in the atrophied areas. A muscle biopsy of her gastrocnemius muscle showed only fat, indicating the reason for the absence of visible atrophy of muscle.

Electromyography demonstrates changes in myotonia atrophica, consisting of a myotonic electrical response, dystrophic muscle potentials on movement and finally polyphasic motor unit responses. In myotonia atrophica the resting muscle shows no electrical potential. The insertion of the needle electrode or tapping with a reflex hammer will induce a burst of motor unit action potentials of varying amplitude at the rate of 200 to 400 per second. This may persist for five to 30 seconds after the needle has come to rest. This is the electrical potential of a myotonic response. On voluntary motion irregular small varying electrical potentials may result, the so-called dystrophic muscle potential. In less involved muscles normal motor unit action potentials may appear on voluntary motion. However, the electrical potentials persist for five to 10 seconds after cessation of contraction. The term polyphasic motor unit response

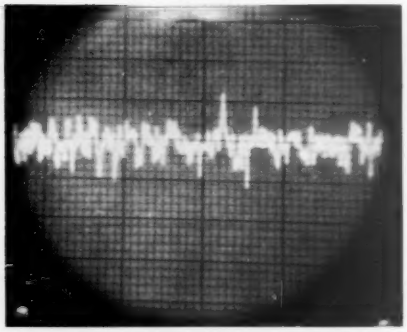

Fig. 1 — Myotonic muscle response to tapping with reflex hammer. Opponens Pollicis muscle of patient W. S. Calibration: vertical, 100 microvolts/inch; horizontal, 30 milliseconds/inch.

refers to a multiphasic, large electrical potential, which may occur on voluntary motion and after mechanical or electrical stimulation. This occurs in areas of muscle which fail to demonstrate a myotonic response and show partial atrophy.

Electromyograms on the mother, M. S., were difficult to obtain because of the paucity of muscle due to the marked fat replacement. An electrical myotonic response could not be demonstrated. However, there were definite polyphasic motor units and a few dystrophic electrical potentials of short duration were elicited. The son, W. S., showed typical myotonic electrical responses on needle and percussion stimulation (fig. 1). These responses lasted from eight to 30 seconds and were found in all muscles except the quadriceps femoris. Sound recordings of the myotonic response were classical, with an initial burst similar to a thunderclap and followed by a dive bomber-like pulsating sound. In addition short runs of dystrophic potential activity during voluntary muscle contraction occurred. This activity was noted in the lower extremities only. The daughter, M. K., showed electromyographic changes similar to those of her brother.

Involvement of the myocardium by the muscular atrophy was suggested in the mother by her electrocardiogram. This showed a PR interval of 0.24 seconds, inverted T waves in standard lead 1 and the precordial leads. The electrocardiogram in the two children showed a PR interval of 0.20 seconds, the upper limits of normal, and bradycardias of 50 and 60 per minute. It was not possible to exclude independent coronary artery disease as a cause of the abnormal electrocardiogram in the mother. Thomasen[1] took electrocardiograms in 12 of his cases and found five with a prolonged PR interval. He also noted bradycardia as a frequent finding. Fisch and Evans[2] reported an autopsied 41-year-old male with myotonia atrophica who had auricular flutter during life and died suddenly. Autopsy showed a persistent thymus and an abnormal myocardium with fibrosis, atrophy, separation of muscle fibers by dense fibrous tissue. Hypertrophied muscle fibers with large rectangular nuclei were scattered about.

Endocrine Changes

The mother, M. S., demonstrated no definite abnormalities of gonadal function. She had normal menstrual periods and gave birth to five children. She stopped menstruating after a hysterectomy for fibroids. No urinary assays of gonadal function were carried out on her. Her daughter, M. K., had a normal menstrual history but had been unable to conceive during her seven years of marriage. Infertility is the common gonadal abnormality found in females with myotonia atrophica. Gonadotropin and estrogen levels were normal in her urine (table 1). The son, W. S., gave a history which indicated normal gonadal function. He stated that he enjoyed normal sexual activity, had been married for seven years and had two daughters. However, examination revealed small soft testes measuring two centimeters in length on the right and three centimeters in length on the left. Examination of his ejaculate showed a total absence of sperm. Urinary gonadotropins and estrogens were normal. Seventeen ketosteroid excretion was definitely diminished (table 1). He demonstrated the common gonadal abnormality seen in myotonia atrophica, that of testicular atrophy and azoospermia.

Thyroid abnormalities were present in M. S., the mother, in the form of thyroid adenomas which were treated by surgical

excision. Subsequently she was given thyroid extract for a presumed hypothyroidism. Her protein bound iodine was normal but her basal metabolic rate was —28 per cent. The daughter, M. K., had a normal serum protein bound iodine and radioactive iodine uptake. Her basal metabolic rate was —13 per cent. She was given triiodothyronine, 25 mcg. daily, for two months without any subjective or objective improvement. The son, W. S., had a normal serum protein bound iodine but had a diminished radioactive iodine uptake at 24 hours. His basal metabolic rate was —15 per cent. He was given thyroid grains, one daily, for several months without effect. No definite thyroid abnormalities could be demonstrated in this family except for the thyroid adenomas (table 2).

Pancreatic dysfunction was present in M. S. in the form of diabetes mellitus which required insulin for control. The children had normal glucose tolerances. Diabetes mellitus is an uncommon complication of myotonia atrophica. In view of the strong family history of diabetes mellitus, its occurrence was probably coincidental. There was no evidence clinically of any adrenal disturbances. However, M. K. and W. S. both showed a slight reduction of the 17 hydroxysteroids. There was also a decrease in 17 ketosteroid excretion by W. S. There was no apparent parathyroid or pituitary disturbance.

Cataracts

M. S., the mother, had noted poor vision for two years due to bilateral cataracts visible on gross inspection. Slit lamp examination demonstrated a fine carpet of dust-like opacities, both in the anterior and posterior subcapsular zones with some larger granules along the posterior suture lines and minimal sclerosis of each nucleus. M. K., the daughter, had no visual complaints. Slit lamp revealed several posterior subcapsular sites of bedewing near the equator of the right lens and similar changes plus a patch of dust-like opacities in the posterior subcapsular region of the left lens. W. S., the son, also had no visual difficulty. Slit lamp examination showed a fine layer of white dots and small grey white flakes with an occasional red, green or blue pinpoint granule in the anterior and posterior cortex midway between the adult nucleus and lens capsule. A lesser number of opacities were seen between this layer and the lens capsule. The axial posterior subcapsular layer had additional opacities along the suture lines resembling an early stellate figure.

Table 1: Endocrine Studies in Myotonia Atrophica

Patient	Buccal Smear	Gonadotropins Mg. of Ovarian Weight per 12 Hrs. of Urine	Estrogens Micrograms per 24 Hours	17-Hydroxysteroids Mg./24 Hours	17-Ketosteroids Mg./24 Hours
Mrs. M. K.	female	12.5	2.7	2.4	6.1
Mr. W. S.	male	33.5	0.8	2.3	4.2
Normal		12-40	1.5-8 female 0.5-1.5 male	3-8	5-15 female 7-25 male

The gonadotropins are concentrated from 24 hour urine by ultrafiltration and assayed in immature female rats of the Sprague-Dowley strain using ovarian weight as the end point. Estrogens were determined from the sodium hydroxide soluble fraction of the chloroform extract of acidified urine and assayed in the immature female rat using the uterine weight as the end point.[13] Results are expressed as microgram equivalents of estradiol benzoate. Urinary 17-ketosteroids were determined by the method of Drekter.[14] Urinary 17-hydroxysteroids were determined by a modified method of Silber and Porter.[15].

Table 2: Thyroid Function in Myotonia Atrophica

Name	BMR	Protein Bound Iodine Micrograms %	Radioactive Iodine Uptake One Hour	Iodine Uptake 24 Hours
Mrs. M. S.	—28%	5.5		
Mrs. M. K.	—13%	5.0	0%	36%
Mr. W. S.	—15%	4.7	0%	7%
Normal	+10% to —10%	3.5 to 8	0 to 5%	15 to 55%

The lens changes in myotonia atrophica are seen best by slit lamp and are considered characteristic of myotonia atrophica by Vogt.[3] Duke-Elder[4] describes the changes as a fine carpet of shiny flakes, white dots and fine dust-like opacities in a sharply defined layer just under the anterior and posterior capsules and the opacities show a play of colors — red, green, and blue. The posterior stellate opacities are seen often according to Fleischer,[5] but are not diagnostic of myotonia atrophica and are probably due to progressive opacification of the lens fibers along the suture line. Meyer[6] found the opacities concentrated along the polar regions. Thomasen believes that the opacities congregate chiefly in the posterior cortex. He found 17 gross cataracts in 101 patients with myotonia atrophica. According to Vogt, all patients with myotonia atrophica will show abnormalities on slit lamp examination with or without gross cataracts.

Cerebral Changes

The mother and two children showed no frank evidence of mental deterioration. There was no memory impairment and judgment appeared normal. They showed a normal quickness of mental response. Initiative seemed to be lacking, as seen in the son who had not been working for two years. He was separated from his wife and family and made no attempt to rejoin them. The daughter failed to follow through on a suggestion for further investigation of her infertility. Their appearance with the slack jaw, open mouth, nasal voice, droopy eyelids gave them a "Simple Simon" appearance. An electroencephalogram taken on the mother was normal.

Thomasen makes a great point of mental deterioration in these patients. He found this to be present in one-third of the patients, with reduced initiative in three-fifths of the patients. Several of his patients were in a hospital for mental defectives. He also stressed their social deterioration and poor working ability.

Discussion

Low basal metabolic rates have been frequently reported in this disease. Lack of clinical evidence of myxedema and lack of response to thyroid hormone also have been observed. The protein bound iodine and radioactive iodine uptake have usually been found normal.[7] The low metabolic rate has been explained by the extensive muscle atrophy, an example of extrathyroidal hypometabolism.[8]

No definite evidence of adrenal insufficiency has been found in myotonia atrophica. According to Thomasen fibrosis and atrophy of the adrenal cortex has been observed at post-mortem, but no clinical evidence of adrenal dysfunction has been reported. Jacobson[8] found 17 hydroxysteroids reduced in two patients, which was similar to our experience. Normal response of ACTH stimulation occurred in his cases.

Pituitary gland dysfunction was not recognized clinically in patients reported here or in the literature. Gonadotropic hormone was found normal in both cases studied here. Thomasen,[1] Jacobson[8] and Clarke[9] report similar findings. However, Clarke found definite elevation of the luteninizing hormone in his two cases.

Pancreatic dysfunction occurs rarely in myotonia atrophica. There has been one patient reported with a sprue-like syndrome.[10] Likewise diabetes mellitus has been rarely reported in myotonia atrophica. Thomasen found no such association in his 101 patients, and found no published reports up to 1948. However, Stanbury[11] reported one patient, and Jacobson reported three cases of diabetes mellitus; Caughey[12] reported decreased glucose tolerance curves in two patients. M. S., the mother, presented this unusual combination of diabetes mellitus and myotonia atrophica.

Attempts have been made to treat these patients with testosterone proprionate, corticotropin and cortisone. Results have been poor or equivocal. The use of Meticorten, thyroid and triiodothyronine were without any definite effect in the three patients reported. Quinine and Pronestyl decrease myotonia, but this is seldom a problem in this disease.

Conclusion

1. A mother, son and daughter with myotonia atrophica are described.

2. The mother showed a late onset of her disease and a masking of atrophy by obesity and coincidental diabetes mellitus.

3. The son and daughter showed low 17 hydroxysteroid excretion; the son excreted subnormal amounts of 17 ketosteroids in the urine.

4. Therapy with Meticorten, thyroid and triiodothyronine produced no beneficial effect.

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Visit not the patient too often, nor remain too long with him, unless the treatment demand it, for it is only the fresh encounter which giveth pleasure.

— ISAAC ISRAELI (Isaac Judeus)

Recent Progress in the Collagen Diseases

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● Medical interest and progress in the field of collagen diseases have grown greatly in the past decade. This has largely been catalyzed by the introduction of steroids both as a treatment and as a research tool. As a result, basic understanding of the biochemical nature of collagen is broadening, diagnostic tests are being purified for greater specificity, treatment methods have been developed for considerably greater effectiveness and rehabilitation technics have been extended with success to the benefit of the chronically disabled patient. The important advances are discussed.

Collagenous or connective tissue is the structural material for the framework of the body. Upon it depends the architectural integrity of each organ, each part, and the human form as a whole (fig. 1). Connective tissue consists of fibroblasts, connective tissue fibers, mast cells and a supporting gel-like ground substance. While the recipe varies from one tissue to another, the basic ingredients are the same, the difference being that of proportions. For example, in the vitreous humor of the eye, the mixture is dominated by a rich mixture of abundant ground substance with a few immature collagenous fibers — this in sharp contrast to muscle tendons in which there is a preponderance of mature, strong connective tissue fibers with only a small measure of gelatinous ground material. The soft subcutaneous tissues; the supporting stroma of lymph nodes, the spleen and other parenchyma; the subsynovial and subserosal stromata similarly are of the same ingredients in the proportions which locally are needed for structure and function. The mother cell of this system, the fibroblast, possesses a versatility only recently appreciated. While its primary offspring is the collagen fiber, it is now apparent that it, also, produces a wide variety of the compounds which are a part of the ground substance. Aside from producing the supporting structural network of connective tissue, the major responsibility of the fibroblast is to respond to injury by migrating to these sites and to extrude in an orderly fashion sufficient collagen for the formation of fibers which in turn will provide the fibrous scar tissue for healing. Brilliant studies of alterations and of factors influencing the metabolic activity of fibroblasts are opening many new windows towards an understanding of the process of aging. Especially is this true in the elucidation of factors which cause vascular sclerosis with its consequent pathophysiologic sequelae. We cannot, today, however, extend ourselves into this fascinating area of natural aging factors. Rather, we must restrict ourselves to an equally enigmatic group of pathologic syndromes of unknown etiology in which the common denominator is an abnormal reaction of the collagenous or connective tissue system (table 1). These so-called Collagen or Connective Tissue Diseases, linked as they are pathologically to a single system, are less firmly aligned etiologically although much evidence has been accumulated to strengthen this latter and to suggest at least an abnormal process of immunity, perhaps autoimmunity, as a common numerator. The most common relegates to this pigeon hole are rheumatoid arthritis, rheumatic fever, disseminated lupus erythematosus, polyarteritis nodosa, scleroderma and dermatomyositis. It is within this restricted frame of reference that our discussion today is directed.

Connective Tissue Research

1. *The Modern Concept of Connective Tissue Disease.* In the absence of any really meaningful evidence as to etiology or etiologies of connective tissue diseases, interest and research have through the years focused on the pathologic changes in connective tissue common to this disease family. Klinge was the first to observe that the diffuse involvement of many organs of the body by rheumatic fever could be correlated only on the basis of the connective tissue

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insult inflicted at each of these sites. He thus postulated that connective tissue as a system might be subject to primary disease and that because of its wide distribution there might result widespread, seemingly unrelated pathology. This concept is today undisputed. With its acceptance, clinicians and pathologists turned with fervor to ferret out and study geographically unrelated pathologic changes in the body which previously had seemed discordant and confusing to the disease format. Rheumatoid arthritis rapidly became recognized as a syndrome with effects far beyond that of synovitis. This propensity for involving a diversity of systems has been documented now for rheumatoid arthritis in almost every body organ and system including the central nervous system. The disseminated pattern of lupus erythematosus originally described as and considered a dermatologic problem took on meaning with identification of its involvement of the heart, kidneys, spleen, etc. Similarly, scleroderma and dermatomyositis assumed perspective as diseases whose pathology was dispersed beyond their dermatologic insults. The medical literature of the 1930's and 40's liberally reflects this fresh awareness and search, as efforts were vigorously directed towards revising concepts and reorienting these diseases into this connective tissue category with its widespread multisystem implications. Though concerned with a smaller group of diseases, this breakthrough was to prove as vital to connective tissue disease as was the identification of bacteria to infectious diseases. For the first time there was delineated a solid base upon which a reliable reference point could be established and from which research pursuits into the unknown could be undertaken without pure hypothesis and without the danger of orbital oblivion.

Table 1: The Major Diseases of Collagen

1. Rheumatic Fever
2. Rheumatoid Arthritis
   Variants:
   - Psorintic Arthritis
   - Still's Disease
   - Arthritis Accompanying Ulcerative Colitis
   - Ankylosing Spondylitis
   - Intermittent Hydrarthrosis
   - Palindromic Rheumatism
   - Reiter's Syndrome
3. Lupus Disseminatus
4. Periarteritis Nodosa
5. Dermatomyositis
6. Scleroderma
7. Degenerative Joint Disease

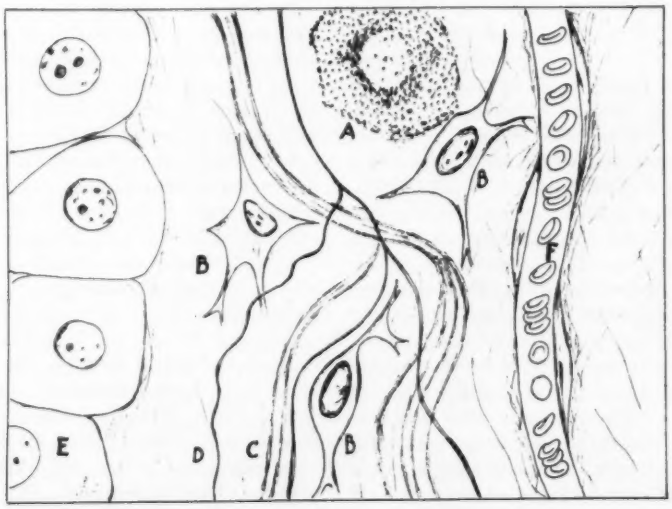


Fig. 1 — Schematic drawing of connective tissue showing a mast cell *(A)*, fibroblasts *(B)*, young collagen fibers *(C)*, and mature connective tissue *(D)* interposed between parenchymal cells *(E)*, and a capillary *(F)*. *(Redrawn from Dorfman.)*

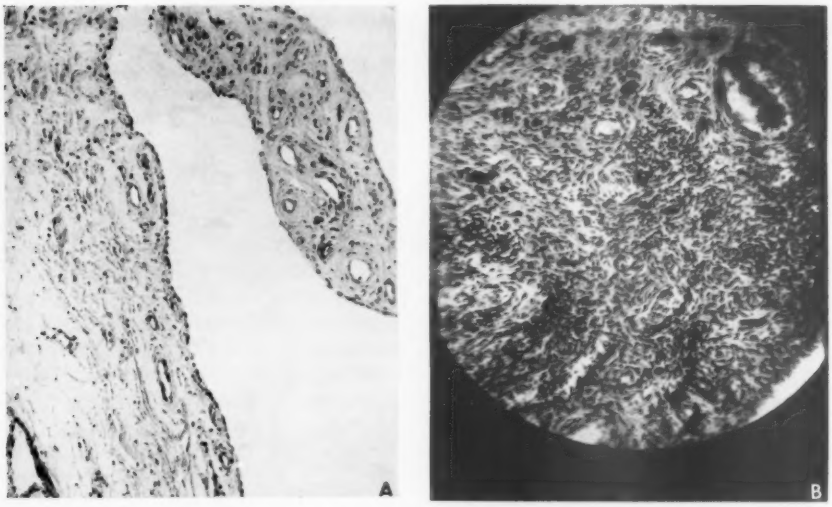


Fig. 2 — (A) Normal synovial tissue showing its loose areolar, highly vascular, and sparsely cellular characteristics. (B) Synovial involvement by rheumatoid arthritis showing diffuse involvement characterized by inflammatory cells and fibroblastic proliferation.

The effects of disease on the connective tissue system are now identified with some clarity despite the fact that causative mechanisms remain obscure. Since a specific tissue can respond to insult in only one pattern, it is not surprising that the pathology of all connective tissue diseases is basically identical. The differences are those of degree. These pathologic changes occur in the following sequence: (1) inflammation to a greater or less extent of the blood vessels and of the connective tissue itself with an appropriate degree of edema in the fibers and interfibrillar ground substance, (2) a proliferative response of all the connective tissue elements — cells, fibers, and ground substance — and finally, (3) maturation of this response as mature fibrotic tissue. This latter stage of organized fibrous tissue may then mechanically limit function — whether in the heart, the joint, the esophagus, etc. — to an extent dependent upon the severity of the scarring and the vital nature of the organ. Thus, the valvulitis and myocarditis of rheumatic fever may heal with scarring that impairs cardiac function. The synovitis of rheumatoid arthritis may progress to fibrosis and joint deformity (fig. 2). The nephrosis of lupus may heal with scarring that produces chronic renal insufficiency. The cutaneous and subcutaneous edema of scleroderma may scar diffusely leaving soft tissues brawny and taut and joints restricted in their mobility. Often the lesions remain chronically active in which cases the connective tissue response is one of continued healing and concomittant continued inflammation — an indication of persistence of the noxious etiologic agent. The severity and acuteness of the connective tissue response may at times reach an explosive force destructive to the part or indeed to the life of the individual. This is especially true of polyarteritis which characteristically involves the connective tissue element of arteries and which may acutely embarrass the nutrient circulation to a part (fig. 3). Where the velocity of the reaction is less accelerated, the healing process may then result in constrictive scarring of the arterial wall which narrows the caliber of the vessel lumen and thus encroaches upon the normal volume of blood flow. When, in either instance, it is an end artery that is involved — a renal, coronary, or cerebral — the result may be a fatal ischemia.

For purity purposes, connective tissue diseases are clinically categorized according to patterns of organic involvement.

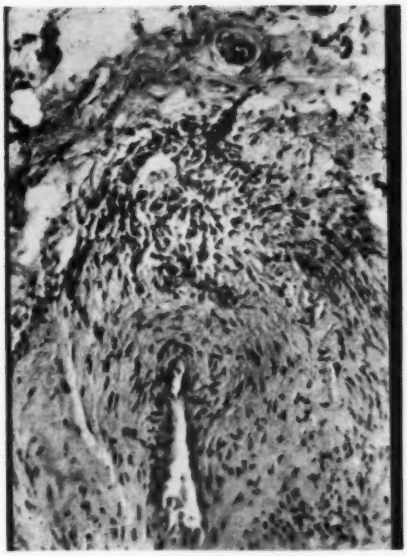


Fig. 3 — Periarteritic necrosis of an arterial wall involving all layers of the vessel.

There is much from a pathologic standpoint, however, to suggest intimate overlaps of pathologic patterns. Many recent necropsy studies have reported finding in single patients the combined features of polyarteritis, rheumatoid arthritis, rheumatic fever and lupus erythematosus. Whether this can be accepted as evidence of a close familial relationship and inbreeding of this group of diseases is questionable. There are some who regard collagen diseases as a spectrum of disorders with the pure syndrome at points along the spectrum but with many shadings or mixed syndromes within the arc. This it would seem is a dangerously thin foothold of assumption since it is based solely upon pathologic response of a tissue with total ignorance of etiology. As previously emphasized, tissues can respond to insult in only one set pattern; it is the intensity of this response which may vary. Hence, until firmer evidence to corroborate etiologic relationships between the so-called connective tissue diseases is forthcoming, it would seem wisest to regard them as disease processes of perhaps widely varying etiologies inflicting damage on the same body system, the connective tissue system. The charred field thus may have been scorched from a bomb or flamed from a match or burned from a careless coal.

2. *Physiology and Biochemistry.* Basic understanding of the biochemistry and physiology of connective tissue attracted little investigative interest until the surge that followed in the wake of cortisone in 1948. As a diffuse structurally monotonous tissue, it lacked the intricacy and physiologic intrigue of more complex parenchymal tissues to be regarded as other than pedestrian. The introduction of steroids fortuitously provided a tool for dramatically modifying inflammatory changes in connective tissue and it was the enigma of this mechanism that stimulated widespread research interest. At the same time, it glaringly spotlighted our primitive knowledge and ignorance of the system beyond normal and pathologic histologic characteristics. The development of the electron microscope at about the same time happily supplied a valuable research tool for satisfying this fresh thirst for explanation.

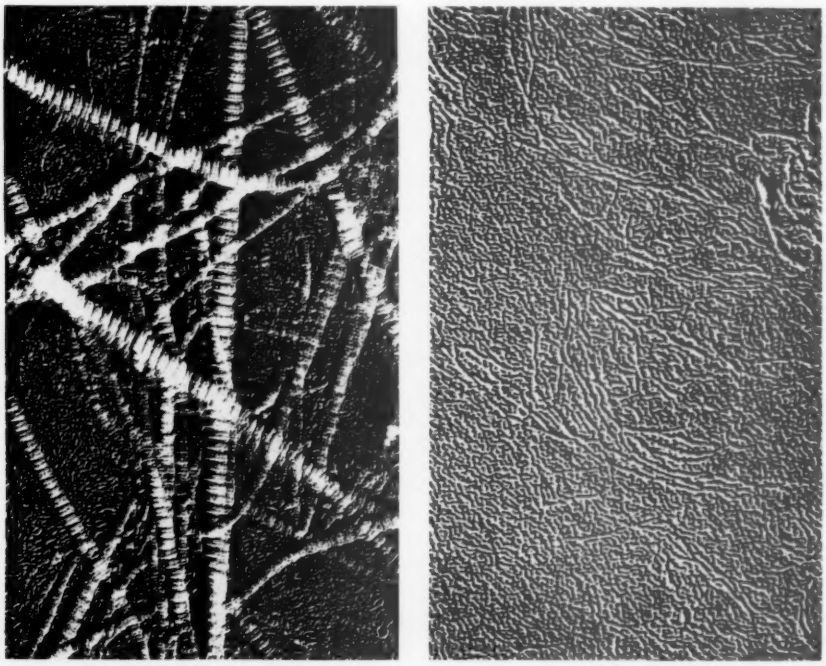
At least eight acid mucopolysaccharides in connective tissues have now been identified occurring either alone or together depending upon the location of the tissue (table 2). Hyaluronic acid is particularly abundant in a number of tissues, especially the synovial fluid. Chondroitin sulfuric Acids A and C are richly distributed in cartilage and, to a less extent, in other tissues. The mast cell is now identified as the manufacturer of heparin, its metachromatic granules being rich in it, and more recently this cell has been identified as one source of the rheumatoid factor which will be discussed later.

Electron microscopy has provided a fascinating window for identifying the structural characteristics of collagen fibers and for observing abnormal changes occurring under various physical and chemical conditions. The collagen molecule is now known to be uniformly rod-shaped with a length of 2500 A and a width of the order of 50 A. Within the proper chemical milieu these molecules aggregate in a precise and orderly fashion to form the collagen fibril. These in turn may combine to form bundles of varying densities and dimensions.

Table 2: Mucopolysaccharides of Connective Tissue (From Ragan)

Heparitin Sulphate	Kerato-Sulphate	Hyaluronate	Chondroitin	Chondroitin Sulphate		
				A	B	C
Aorta All Blood Vessels?	Cornea Nucleus pulposus Bone Adult Cartilage	Synovial fluid Umbilical cord Skin Ligaments Aorta Tendon Almost all connective tissue	Cornea	Cartilage Bone Aorta Cornea	Skin Tendon Heart valves Aorta Yellow ligaments	Nucleus pulposus Cartilage Bone Tendon Embryonal tissue

Long before the availability of electronic microscopy for direct visualization, it was known that collagen fibers would dissolve when exposed to a dilute acid solution and that the fibers would reprecipitate in an orderly structural fashion by the simple process of neutralization. This phenomenon can now be visualized microscopically (fig. 4). The amazing faculty of the fiber to dissolve and lose all structural form simply as a result of exposure to acid and to reassemble itself into a functional tissue upon neutralization of the media has more than any other discovery stimulated speculation as to the role of this phenomenon in disease processes. That this is not an enzymatic effect is shown by the fact that the phenomenon is reproducible when *pure* extracts of collagen are used. This property is unique to collagen tissue and is not duplicable with elastic fibers. It is of interest that aging of collagen fibers modifies the phenomenon and that solubility decreases with age. The implications of the latter in the understanding of diseases of aging are intriguing teasers.



(A) (B)

Fig. 4 — *(A)* Collagen fibers showing orderly architectural arrangement. *(B)* After exposure to a dilute acid solution the collagen molecules have disassociated themselves into solution. *(From Bauer.)*

Another phenomenon of interest which was first observed quite by accident is the effect of the proteolytic enzyme, papain, on cartilage. When papain is injected intravenously in a rabbit, the animal's ears collapse. This is due to loss of cartilage matrix, the result of the disappearance of chondroitin sulfate, the latter apparently released by the proteolytic enzyme, papain. This rather rapid effect, with loss of rigidity and structural form of the cartilage ordinarily reverses itself within two or three days. The cartilage histologically becomes reconstituted, regains form and rigidity, and the rabbit's ears reassume their normal upright positions. This accidental observation in itself is illuminating evidence of the relative nature of the integrity of cartilage. A fascinating further observation is that the administration of cortisone to the rabbit injected with papain will block the recovery of cartilage to its normal form and in such animals the ears remain limp.

3. *Abnormal Macroglobulins* (table 3). One of the most rewarding probes into the unknown of rheumatic diseases has been the identification and study of macroglobulins. As familiarity with these has increased, diagnostic tests have been developed and broad new concepts of the basic pathologic mechanisms involved have evolved. The latter in turn when clarified may well provide a sound base from which the etiologic wall may be cracked.

Macroglobulins is a general descriptive phrase applied to those large serum globulins that have a sedimentation coefficient exceeding 14. Their molecular weights obviously are heavy. Macroglobulins normally are present in small amounts in serum, three per cent or less of serum proteins being of such size. These may be of the alpha-2, beta, or gamma globulin types. Macro gamma globulins in normal sera are mostly antibodies. Except, however, in disease macro beta globulins occur in only trace quantities. Abnormal macroglobulins are found in certain diseases such as syphilis, kala-azar, sarcoidosis, and some diseases of the liver. More recently, they have been consistently identified in rheumatoid arthritis, and considerable evidence is collecting to corroborate their presence to a less extent in other connective tissue diseases. It is these macroglobulins which constitute the so-called "rheumatoid factor" and which are the basis for the many variations of the agglutination test used now in the diagnosis of rheumatoid arthritis (table 4). The most recent modifications of this agglutination test are positive in greater than 90 per cent of cases of rheumatoid arthritis with less than five per cent positives in the controls.

Table 3: Macroglobulins

Normal	Antibodies, such as:
	Isoagglutinins
	Heterophiles
	Rh Antibody
Pathologic	Macroglobulinemia
	Kala Azar
	Syphilis
	Sarcoidosis
	Liver Disease
	Cold Hemoglobinuria
	Rheumatoid Arthritis
	Other Connective Tissue Diseases

The rheumatoid factor macroglobulin has a sedimentation coefficient of 19S and a molecular weight in the range of one million (fig. 5). It is rich in carbohydrate, has a mobility of gamma globulin and has antigenic properties of a specific antibody. If, however, the rheumatoid macroglobulin is truly an antibody, the antigen is yet unidentified. The evidence, however, supports an hypothesis for such a system. The 19S rheumatoid macroglobulin exists as a complex combined with the small 7S gamma globulin and with no other protein so far as is known. This would suggest an antigen antibody relationship between the 7S and 19S gamma globulins — actually an autoimmune reaction. This bound gamma globulin complex of which the 19S macroglobulin is a component is to be found in the fibrinoid changes of rheumatoid arthritis, in the connective tissue and in certain inflammatory cells of the exudate. More recently the rheumatoid factor macroglobulin has been demonstrated consistently to be present in the plasma cells of the synovia, in the plasma cells of rheumatoid nodules, and in the plasma cells, the Russell body cells, and the germinal center cells of lymph nodes of

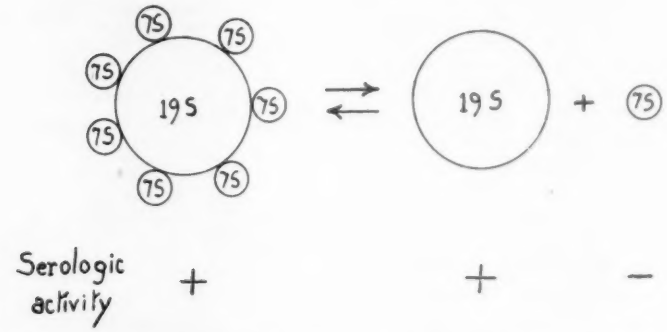


Fig. 5 — A schematic interpretation of the rheumatoid antigen-antibody reaction. *(From Kunkel.)*

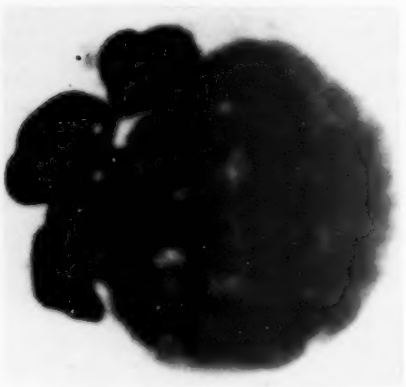


Fig. 6 — A lupus erythematosus cell. *(Courtesy of Dr. Morris Ziff.)*

patients with rheumatoid arthritis. These specific cells are now considered to be the production centers for the rheumatoid macroglobulin and adds considerable weight to the proposal that it is an antibody.

4. *Autoimmune Mechanism.* Another break through towards an understanding of the basic pathophysiologic mechanisms in rheumatic disease has occurred in disseminated lupus erythematosus. As with the rheumatoid factor of rheumatoid arthritis, research has concentrated on elucidation of the immunologic aspects of the so-called LE factor. While recognition of the lupus erythematosus cell dates back over a decade to Hargraves, understanding of the mechanism responsible for the phenomenon has accrued slowly and is still far from completely clarified.

In the sera of patients with disseminated lupus erythematosus, there is found an abnormal protein which is an antibody to nucleoprotein. The lupus erythematosus phenomenon results from a typical antigen-antibody reaction in which the gamma globulin of the patient's serum interacts with the desoxyribonucleoprotein of the nuclei of cells. This antigen-antibody reaction causes nuclear swelling in the cell, loss of normal chromatin, and loss of cytoplasm. The altered nucleus, which takes on new and characteristic staining properties, is then phagocytized (fig. 6). It is the leucocyte which contains this phagocytized homogeneous nuclear material that constitutes the lupus erythematosus cell. This immunologic reaction can be consistently demonstrated by many technics such as complement fixation tests, fluorescent antibody methods, latex particle agglutinations, etc. Complement is necessary for the successful interaction of the system. More recently, evidence has accumulated to support the fact that instead of a single anti-nucleoprotein factor, there are probably a number of other factors reactive with various constituents of the cell. While the significance of these multiple antigenic systems is not clearly known, it is of considerable speculative interest that the nucleoprotein reactions are not species specific while the cytoplasmic reactants are specific. It is also of interest that one investigator recently has demonstrated a delayed skin reaction by patients to their own leukocytic extracts. The lupus erythematosus

factor by all measures is an antibody gamma globulin. Though consistent in lupus, it is not exclusive to this disease. Similar reactions are observed in some patients with rheumatoid arthritis, dermatomyositis, and scleroderma and in occasional patients with liver disease. While there is some difference in these non-lupus reactions, their significance is not known.

As more and more evidence has accumulated supporting antigen-antibody systems in rheumatic diseases, a common denominator for the first time is evolving. While this, of course, may represent only a common relationship in pattern of pathologic reaction without etiologic implications, it has nonetheless stimulated intensive efforts to further explore the unknown. In this endeavor, a research tool of recent development and invaluable assistance has been the use of the fluorescent antibody technic. This is a histochemical method of study in which antibodies labelled with fluorescin are used in the staining of fresh tissue sections. By using various labelled antibodies, specific antigens can be localized by identifying the fluorescent antibody under ultraviolet light. This technic has been particularly informative in the rheumatic diseases in which the antigen-antibody systems already described result in bound gamma globulins. By interjecting the appropriate fluorescent antibody it has been possible to detect both the locations and the extent of antigens peculiar to each of the diseases. It was through such a technic that the rheumatoid factor macroglobulin was identified in plasma cells and the cells of lymph nodes as reported above. The wire loop lesion in the renal glomerular tuft, the onion-skin changes of splenic arteries, and the "inclusions bodies", all of which have long been pathologic hallmarks of disseminated lupus, have now been shown fluorescently to be rich in deposits of bound gamma globulin. Similar deposits have been shown to exist in the necrotizing angiitis of periarteritis nodosa, within the fibrinoid changes and the Aschoff bodies of the myocardial lesions of rheumatic fever, and the subcutaneous nodules and within the synovia of patients with rheumatoid arthritis. That these concentrated deposits of bound gamma globulin represent specific and significant antigen-antibody reactions is emphasized by the negative findings in other renal and myocardial diseases of different etiologies.

Use of the fluorescent technic has now been extended to the search for autoimmune bodies in the sera of patients with rheumatic diseases. By this method autoantibodies have been demonstrated against the nuclear constituents of cells thus corroborating the hypothesis that the lupus erythematosus phenomenon is

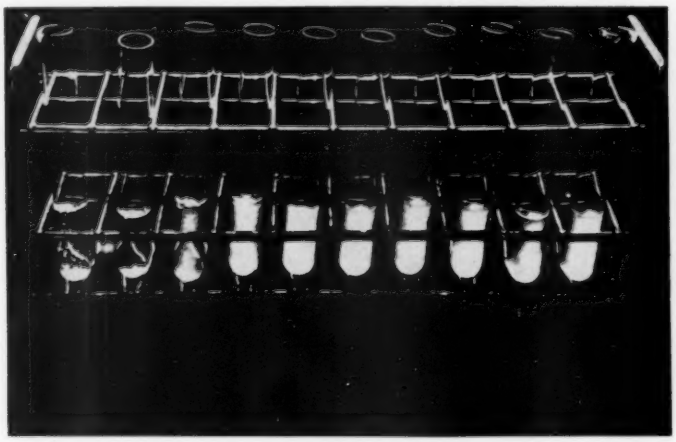


Fig. 7 — A rheumatoid agglutination system fluorescently tagged showing agglutination in the three tubes on the left. *(Courtesy of Dr. Morris Ziff.)*

an autodestructive effect on cell constituents. Similarly, autoantibodies to myocardial sarcoplasm have also been identified. Most recently it has been shown that serum rich in rheumatoid factor is profoundly reactive with the synovial tissue of patients with rheumatoid arthritis (fig. 7). These newer avenues for research are thus fruitful in promises if at times confusing in interpretation.

5. *Pathology.* In the renewed search for the etiologic causes of the connective tissue diseases, a much clearer concept of the basic pathologic processes at work has crystallized and accordingly a more meaningful classification of this diffuse group of syndromes. Further, the identification of the abnormal proteins just discussed and their useful application in specific laboratory tests has strengthened the reliability and extended the breadth of this new perspective.

In the past, the pathology of rheumatoid arthritis has been thought of in terms of the granulomatous lesion typically seen in the synovial membrane and in subcutaneous nodules; that of disseminated lupus erythematosus in terms of wire loop renal lesions, onion-skinning of splenic arteries, and fibrinoid changes in connective tissue; and that of periarteritis as acute necrosis of arteries. The relationship between such varied pathologic syndromes was difficult to accept without concern, and the unity of the family was more one of convenience than of geneology, the only common tie being pathologic insult to the connective tissue system. In the past decade intensive efforts have been directed towards studying these variable pathologic patterns of the connective tissue diseases in a search for more significant evidence of relationship. It now appears that the common link is the fact that the primary lesion is a vasculitis; the variations in the syndromes result thus from differences in extent, location, and degree of vascular injury. This was first demonstrated in lupus erythematosus in which it was shown that the initial lesion is an acute vasculitis with edema of the vessel wall. Secondarily, then, impairment of blood flow through the vessel lumen may result in ischemic necrosis of the tissue distal to the lesion and nutritionally dependent upon it. As a further complication, the vessel in its reparative efforts, may be-

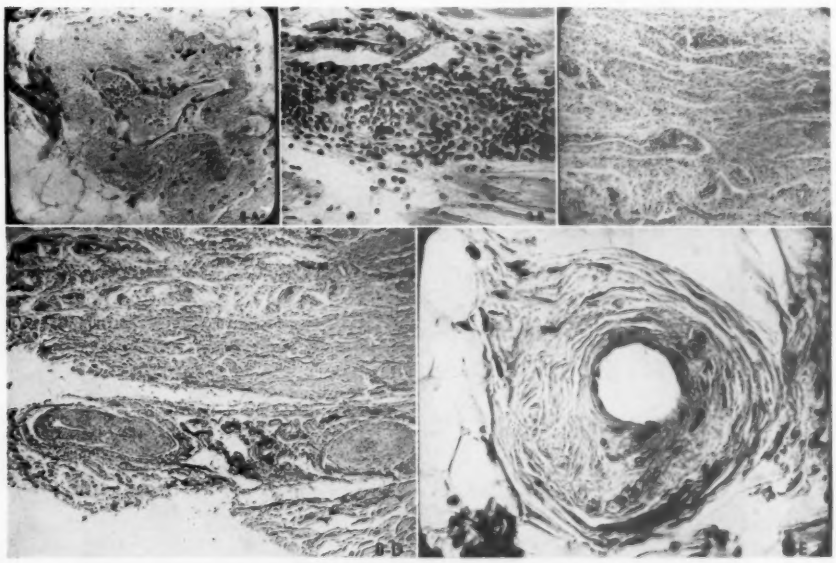


Fig. 8 — *(A)* Early severe vascular edema. *(B)* Secondary acute inflammatory reaction within and about a vessel. *(C)* Diffuse vasculitis plus edema and inflammation of the connective tissue. *(D)* Fibrous healing, the end stage of the acute inflammatory process. *(E)* High power of a healed sclerotic small vessel.

come sclerotically scarred and thus to a greater or less degree permanently impair its patency. Thus, the explanation for the wire loop glomerular lesions, the sclerotic onion-skin arteritis, etc. — all residual pathological stigmata (fig. 8).

Clarification of the pathologic pattern in rheumatoid arthritis was less readily accomplished. The lesions of this disease though most pronounced in joints may be found in many widely unrelated systems. In the serosal membranes such as the pleura and pericardium, it produces a diffuse inflammatory and exudative process. In loose connective tissue, especially in the synovia and in subcutaneous tissues, its lesion is a highly cellular granuloma with central necrosis. In the vascular system, it may produce acute inflammation of large or small vessels on either side of the tree. It was interest in the latter lesions and the accumulation of evidence of a high incidence of arteritis in rheumatoid arthritis that redirected pathologists to search for a vascular lesion as the basic cause of the granuloma. It is now well established that the primary pathologic lesion of rheumatoid arthritis is a vasculitis. This, as in lupus, results in edema of the vessel, endothelial thickening, restriction of blood flow, ischemic necrosis, and a secondary inflammatory response of cellular infiltration and fibroblastic proliferation. This sequence is the same in the synovia, in the subcutaneous nodule, and elsewhere. Whether there is any specificity which might explain the localization of lesions is not known. It is of interest, however, that the lesions of rheumatoid arthritis occur most often in areas and tissues subjected to greatest mechanical stresses where abnormalities of physiology might be expected to be most acutely manifested.

6. *Classification.* The identification of specific globulins in various of the connective tissue diseases and the application of their immunologic properties in the development of special diagnostic tests, specifically the lupus erythematosus cell test and the sheep erythrocyte agglutination test and its variants, has provided informative tools for settling longstanding controversies over classification (table 5). Rheumatoid spondylitis, for example, has always posed a major problem of disagreement. Despite many major differences in sex incidence, hereditary transmission, prognosis, and response to therapy, Americans have regarded it as a spinal counterpart of peripheral

Table 4: Tests Results in Connective Tissue Disease (From Hall, Bayles, and Bardawil)

	Fluorescent Antibody Test	Latex Test	Lupus Erythematosus Preparation
Rheumatoid Arthritis	Negative	Positive	Negative
Disseminated Lupus Erythematosus	Positive	Negative	Positive
Scleroderma	Positive	Negative	Negative
Polyarteritis Nodosa	Equivocal	Equivocal	Negative
Dermatomyositis	Equivocal	Equivocal	Negative

Table 5: Sheep Erythrocyte Agglutination Inhibition Tests in Various Arthritides[5]

Disease	No. of Patients	No. Positive	Per Cent Positive
Rheumatoid Arthritis	140	135	96.4
Juvenile Rheumatoid Arthritis (Children only)	24	23	95.8
Ankylosing Spondylitis	119	1	0.8
Psoriatic Arthritis	76	1	1.3
Arthritis Accompanying Ulcerative Colitis	22	0	0.0
Reiter's Disease	41	1	2.4
Systemic Lupus Erythematosus	18	5	28.0
Controls*	321	12	3.7

*Osteoarthritis, gout, rheumatic fever, miscellaneous nonrheumatic diseases and normal individuals.

rheumatoid arthritis. This has largely been because of the frequency of peripheral joint involvement accompanying the spondylitis. European rheumatologists, on the other hand, have vigorously insisted that spondylitis is an entity apart from rheumatoid arthritis. Major support for this latter has now accumulated from results of the rheumatoid agglutination tests. While these are positive in more than 95 per cent of patients with rheumatoid arthritis, these tests are consistently negative in spondylitis. The arthritis of ulcerative colitis, the typical arthritis associated with psoriasis, and that a part of Reiter's Syndrome are now similarly isolated into separate classification categories by virtue of negative reactivity of the agglutination test. Conversely, Still's Disease which by some has been regarded as an entity separate from adult rheumatoid arthritis is now shown to produce positive rheumatoid agglutination results in the same ratio as rheumatoid arthritis; it thus must be considered as the juvenile equivalent and basically the same disease process.

Table 6: Action of Rheumatic Disease Drugs

Specific	Analgesic	Anti-Inflammatory	Unknown
0	Salicylates	Salicylates (?)	Gold Salts
	Phenylbutazone	Phenylbutazone (?)	Chloroquine
		Corticosteroids	
		ACTH	

Table 7: Rehabilitation Evaluation of the Arthritic Patient

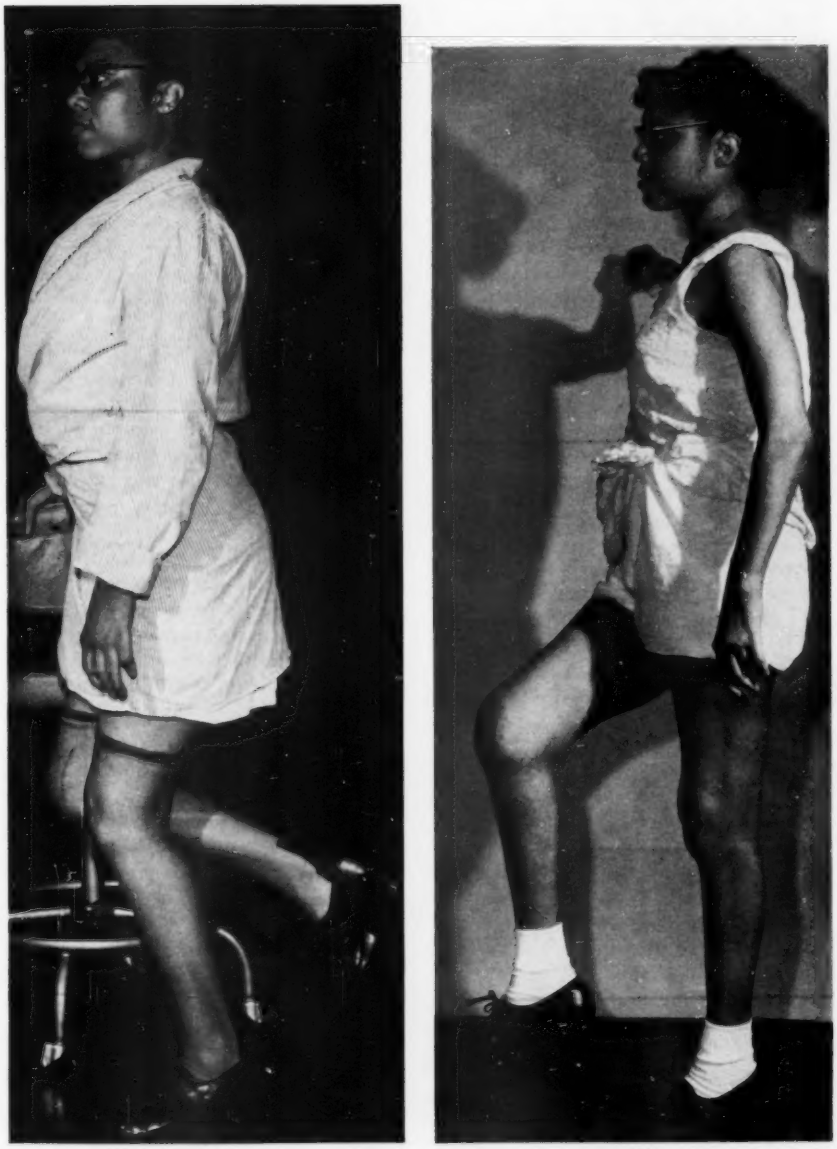
Medical History Physical Examination Specialist Consultations Laboratory Examinations	▸ Diagnosis and Physical Prognosis
Muscle Test Joint Range of Motion Speech and Hearing Evaluation Activities of Daily Living	▸ Functional Capacity and Potential
Psychological Testing Social Survey Vocational Testing	▸ Economic Potential
	Rehabilitation Potential

The TOTAL PATIENT must be evaluated Socially, Psychologically, and Vocationally as well as Medically and Functionally before a realistic goal may be established for the individual.

Treatment

Despite these considerable inroads towards fuller understanding of the connective tissue diseases, little as yet is applicable in a practical sense to the treatment of the diseases. We are, thus, clinically still contending with diseases of unknown etiology, and management terrains remain largely empirical ones. The possible exception to this is rheumatic fever, the disease most closely identified etiologically and most effectively treated on the basis of its beta hemolytic streptococcus relationship. Even here, however, it is the prophylactic phase of management which is most effective in contrast to the still troublesome acute phase. Drugs (table 6) for the treatment of connective tissue diseases are of three types: (1) analgesic, (2) those that suppress inflammation, and (3) those which for reasons unknown alter the course of the diseases in a significant percentage of cases. The corticosteroids are representative of the anti-inflammatory agents used therapeutically. Their supressive effect on inflammation is totally non-specific without influence on causative mechanisms. Their value is solely as a blocking agent which in varying degrees of effectiveness prevents the connective tissue reacting in a protective effort against the unknown noxious agent. That the inflammatory reaction may proceed unchecked against the persisting noxious stimulus and produce self damage and destruction has previously been indicated. Dampening of this response with corticosteroids is thus of some protective value, non-specific though it may be. Among drugs of therapeutic value in deterring the course of some connective tissue diseases are gold, antimalarials, and phenylbutazone. In none of these is a mechanism of action known, nor are responses of the diseases consistent to their use.

With the exceptions of the above addenda to our therapeutic armamentarium, management of connective tissue diseases remains basically unchanged from that of past decades. Good management is one of conservatism aimed at building normal resistive resources of the body, hopefully, to a level of self-cure, while at the same time using medications for analgesia and a sound physical medical program for the prevention of joint



(A) (B)

Fig. 9 — Pre- and post-operative posterior capsulotomy surgical releases for flexion contractures of knees.

deformities and muscular atrophy. The wisdom of this time-tested formula as the basic therapeutic approach has repeatedly and disastrously been emphasized by the frequent and at times catastrophic complications from steroids, gold, and other empirical drugs. These latter, therefore, are truly as described, addenda to the basic treatment regimen of rest, salicylates, a proper diet, and a judiciously prescribed physical medical program of therapeutic exercise. This in no measure is intended to negate the value of steroids, gold, etc., as therapeutic tools which when properly used are often life-saving in quality. Rather, it cannot be overemphasized that these are two-edged swords with potentially grave undesirable effects and that they should be used with cautious decision and deep respect for the hazards to which one is subjecting his patient.

Reliance upon conservative measures and especially upon good physical medical measures in the management of the connective tissue disease is today re-emphasized rather than de-emphasized and the importance of this will continue in top priority until that fortunate day when the riddles have been resolved, and cures are within our currently ineffective hands.

Rehabilitation (Table 7)

Finally, let us consider one area in which there has shone new light and hope — that is, in the rehabilitation of persons unfortunate enough to have become disabled cripples from these diseases. With the maturing of Rehabilitation as a medical specialty for the comprehensive treatment of chronic disability, it was inevitable that research would eventually focus on the rheumatic diseases; inevitable, because, of all physically disabling diseases, these afflict the greatest numbers of our population; inevitable, because the high morbidity and the low mortality of these diseases arm them with a mounting threat to our social and economic structure.

The problems of chronic disability extend far beyond purely medical limits. Among these none are more profound and complex than in rheumatic disease, for in most cases they are progressive with worsening disability and with concomittant pain. Repeated studies have now conclusively demonstrated that many patients disabled by rheumatic disease can be rehabilitated and restored to useful lives. Treatment to be successful must be broadly directed towards the total patient and not only towards medical management. The latter is, of course, primary in importance, but equally as crucial is a treatment program directed towards reversal of contractures and muscle weaknesses, dynamic social service to resolve social restrictions limiting independence, psychologic care, and accurate assessment of vocational skills which may be used in employment compatible with the residual physical disability. Obviously, the successful resolution of the crippled rheumatic's problem demands skills beyond those of a single physician. He relies therefore upon a team of specialists each of whom is professionally capable of assisting with an appropriate facet of the disability. It is only through such joint efforts of the physician, the physical and occupational therapists, the social worker, the psychologist and vocational counselor that all walls of the problem can be demolished and the patient freed of his restrictions.

In instances of irreversible deformity which present impasses to physical independence, orthopedic surgeons are now aiding invaluably with reconstructive surgery (fig. 9). Long-standing conservatism and even disinterest in this type of chronic problem is slowly but encouragingly being replaced by vigorous optimism among a new generation of orthopedists oriented more to the looming challenge of chronic diseases than to that of acute trauma. Technical surgical skill is a prerequisite to a favorable result, but it has been repeatedly emphasized that of equal importance to surgical technic is the intelligent prescription of pre- and post-operative therapeutic exercise programs and the diligent supervision of these.

The most difficult hurdle in the return of the rheumatic disease cripple to self-sufficient living is competitive job place-

ment. Obstacles to travel, physical limitations, the competitive labor market, the esthetic stigmata of deformity are but a few of the blocks encountered. Even this, however, is by no means insoluble. Recent demonstration projects have eloquently rebutted objections to employment of the arthritic. With intelligent vocational evaluation, seasoning, and training, the average arthritic can be job placed without preferential considerations. In one remarkable study alone, 40 per cent of otherwise "unemployables" were job placed. Further, it was shown that disease exacerbations, pain, accident proneness, lack of motivation, and absenteeism did not mar the employment records of these persons.

This, then in brief, is a survey of recent advances in our understanding of connective tissue and connective tissue diseases. To say the least, it is an oversimplification of complexities. Nonetheless, it is a bright picture of progress accomplished and being accomplished. [4]

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Teaching of Rehabilitation in a Medical School

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● This paper reviews the growth of departments of physical medicine and rehabilitation, the teaching of this specialty, and the nature of the medical school faculty which decides how the teaching of physical medicine and rehabilitation is conducted. For adequate teaching a service area for demonstration of the utilization of team personnel in a patient-centered approach is necessary. Presently-utilized areas, aspects of curriculum planning and what medical students should be taught concerning rehabilitation are discussed. Experience of the author and his associates have convinced them that it is illogical to assume that exposure to concepts of rehabilitation will result in a whole-hearted acceptance of them by all students. In each class, however, they have found that from 10 to 15 per cent of the students have exhibited much above average interest in the problem of chronic illness and the comprehensive care that is involved in such illness. This may appear to be a small number, yet if one considers that there probably are not more than five per cent of the physicians in the United States who are enthusiastically interested in the problem of rehabilitation, increasing this group to 10 to 15 per cent in our schools of medicine will create a large enough nucleus of physicians for the future who can meet the challenges that lie ahead.

Rehabilitation may be defined as a complex of the varying processes which are needed to restore an individual to a position of utilizing his maximum physical, psychologic, social and economic potentials. The multiplicity and extent of the services required to achieve this process are directly related to the magnitude of the disability. In some instances only a few services are needed; in others, many. Viewed from this perspective, rehabilitation becomes a concept of patient care rather than a defined body of knowledge, yet an increasing body of knowledge is needed to make the concept a reality.

Although to some individuals rehabilitation is needed chiefly in the third phase of medicine, after preventive and curative measures have been employed, it is essential that the initial steps in rehabilitation be instituted early if the patient is to avoid the deleterious after-effects of early neglect. This should be the responsibility of the physician. This is true of all physicians, but as in other phases of medical care, it becomes more clearly the responsibility of the physician who assumes the role of personal physician to the patient. The advent of more and more specialization should not destroy this usefulness of the patient-physician relationship.

The philosophy embodied in the concept of rehabilitation should flow naturally from all medical care in that it is patient-centered rather than disease-centered. That such is not the case is manifested by many surveys, by the reaction of the public which receives medical services, and by any assessment of the end results in chronic disease care. True, there are many physicians who try to the best of their ability to give the patient comprehensive care. Unfortunately, it is not always within the compass of each physician to provide by himself more than a portion of the total care which the patient may require.

As a result, historically, there have arisen many co-professional groups or auxiliary groups, some of which have been physician-initiated, others which have been started by well-motivated men and women outside of the medical profession who were primarily concerned about improving the lot of their fellow-men. As time has gone on, these groups have organized themselves into various disciplines with a more or less defined body of knowledge and responsibility. In some instances their activities may have challenged the physician in his traditional role with the patient, often by default on the part of the physician. In his defense, however, there has not been in the medical curriculum adequate time for proper orientation of the physician to his responsibility for leadership in meeting the comprehensive needs of the patient.

In the past decade there has been an awakening of educators to this need of more physicians trained in concepts of rehabilitation and many medical schools have encouraged introduction of concepts

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Fig. 1 — The "team" as seen by the patient.

of comprehensive care, increased training in the relations with and utilization of co-professional personnel, and some education in the basic technics of rehabilitation used by the physician as well as some co-professional groups.

The innovator in medical education concerned with rehabilitation has had to take much knowledge which has existed in part in many disciplines, redesign it in the light of newer knowledge and technics, and then attempt to re-enter the curriculum. This often has resulted in jurisdictional disputes which have obscured the basic objectives in medical education, namely, to provide within the framework of four years enough basic knowledge of morphology and function of the human body, enough understanding of human personality and mind and its interaction with other individuals so that the student might, with appropriate postgraduate work, either elect to pursue his career primarily of rendering service to patients or doing research, or better still, combining both.

To achieve these goals it is necessary to utilize the members of the faculty who have an "enthusiastic attitude" concerning rehabilitation. These come from the various disciplines of physical medicine, internal medicine, neurology, pediatrics, orthopedics, otolaryngology, surgery and psychiatry. This enthusiastic group of teachers gradually can inculcate in other members of the faculty first a passive acceptance of rehabilitation concepts, then later an active participation and enthusiasm. The same spread of a concept in our teaching program has occurred in the last 50 years in two other areas, namely, preventive medicine and psychiatry. Any concept which embodies both the old and the new requires conflict and the passage of time to define the necessity for its existence.

Responsibility for the Teaching of Rehabilitation

Since the establishment of a Department of Physical Medicine as early as 1929, there has been a significant increase in such departments in medical schools across the country. Department status has been achieved by 18 of the 82 medical schools in the United States, and one of 12 medical schools in Canada, according to the latest Directory of the Association of American Medical Colleges.[1] Four of these schools designate the departments "rehabilitation medicine," three are designated "physical medicine," whereas the rest are "physical medicine and rehabilitation" departments. Undoubtedly in other medical schools the physical medicine and rehabilitation areas have a somewhat autonomous status within the jurisdiction of major departments. The establishment of such departments in some instances represents the pressure of strong, enthusiastic leaders who have convinced curriculum committees of the need of departmental status for their specialty rather than an acceptance by the traditional areas of instruction in the medical school.

On the other hand, in many other medical schools there is a strong con-

viction that the teaching of rehabilitation is not the province of any one department, but rather should be coordinated on an interdepartmental basis. In many such institutions the leadership has come in varied fashion from different specialties. The present trend in medical education is not to create more departments but rather to unify and integrate those which are already in existence.

Johnson, Worden and Burk[2] have expressed themselves strongly that the growth of physical medicine and rehabilitation depends on an increasing supply of well-trained physiatrists. I agree that a physician trained in a standardized well-rounded residency program as they describe it can provide the leadership needed in the teaching of rehabilitation in a medical school. In most medical schools such researchers must first run the gamut of the administrators, researchers, and teachers of other fields. A re-evaluation of many present residency programs is needed so that more such leaders can be produced.

During the past five years the success of our teaching program in rehabilitation, under the jurisdiction of the Department of Medicine, indicates the desirability of continuing it in this particular department. The Department of Medicine occupies nearly a fourth of the total curriculum hours of the School of Medicine and is the largest single department, thereby coming into greater contact with the students than any other area.

The leadership of the Department of Medicine assures the cooperation of other areas in the curriculum and among the faculty. It would seem unwise on the part of any medical faculty to place arbitrarily the responsibility for the teaching of as broad a concept as rehabilitation exclusively in the hands of one specialty. This tends to create in the student's mind the idea that rehabilitation is a specialty rather than a concept of care which should be recognized by all physicians.

Leadership in each school for disseminating concepts of rehabilitation must be secured from those members of the faculty who are enthusiastic about its value in medical education. As Rusk[3] has pointed out, "the primary factor is not the specialty board to which the physician is credited but rather the answer that can honestly be given to these two questions:

'1. Does the individual have personal characteristics which motivate him to work with and understand disabled persons?

'2. Does he have the necessary training in the specialized skills and techniques of rehabilitation?

'If the answer is "yes," then little does it matter whether he is a physiatrist, internist, orthopedist, neurologist, or a specialist of any other sort'." Above all, the leadership must be such as can secure cooperation with the major departmental areas in which rehabilitation can be best demonstrated and taught.

There are some who feel that competence in rehabilitation skills alone is adequate to secure acceptance of rehabilitation's place in medical education. I do not believe this is so since the nature of a medical faculty may militate against its ready acceptance.

Nature of the Faculty

The medical school is a complex professional community composed primarily of three groups, the teachers, the administrators and the researchers. Ekstein and Wallerstein[4] have aptly described the self-image each of these groups has built.

The teachers, the largest group, often feel that they are belittled and relegated to the last position. They view themselves as the proud carriers of classical tradition, who in dedicated fashion give of themselves to their students. They may feel, nevertheless (and with some cause), that their efforts are not adequately appreciated, particularly when faculty position and salary seem to be dictated by the amount of research produced and number of articles published. This group often feels that there are more important concepts and factual material to be taught before allotting curriculum time to rehabilitation.

The administrators, on the other hand, view themselves as those who bring order into the chaos where the others move. They feel they provide the leadership for

Fig. 2 — The "team" as seen by the medical student.

an irresponsible group of individuals who can't see the whole because of their narrow departmental interests. Many times, however, they feel looked down upon as hucksters who must sell the program to the public, raise the funds, and who must constantly make things bigger even if they aren't any better. This group because of their broad point of view have been usually sympathetic to the spread of rehabilitation concepts, but must temper their support because of the presence of strong traditional groups in the faculty.

The researchers, the third group, like to stress their dedication to science and their inherent right of academic freedom. They see themselves as the only true carriers of progress and the continued testers of dogma. Often they view the teachers as obstacles to scientific advances, the administrators as barriers to creative work. They too have their moments of dejection when others fail to appreciate their lofty attitudes of better serving mankind and science, or when granting agencies fail to meet their needs, or try to control the direction of their research. This group, unless approached correctly, is apt to see in rehabilitation a point of view which does not lend itself to scientific study, but which because of easy publicity can gain much public support. Although there has been excellent research in the area of physical medicine, there is much need for additional research in the broad field of rehabilitation.

Thus rehabilitation must be taught in the atmosphere of the constant struggle within the medical faculty, which includes still further the rivalry caused by differences in university degrees, salary scales, and faculty status.

Some schools have tried to attain these goals by loosely coordinating the efforts of various departments with token efforts on the part of each. In such instances, the concept makes very little headway. Kant once said, "I do not intend to teach you the philosophy, but how to philosophize"; so too, in rehabilitation it is not enough to teach the student a concept of rehabilitation, one must also teach him to rehabilitate the patient. This requires leadership provided in one department or area.

Each medical school varies from another in the strengths of various departments. Administrators would do well to build their teaching of rehabilitation on those segments of the faculty who feel that a comprehensive care philosophy is a requirement for the graduate. A philosophy, however, is not enough.

Need of a Demonstration Area

This raises the question of the need of a service area for demonstration of the utilization of team personnel in a patient-centered approach. This appears to be absolutely essential in the teaching of rehabilitation. In our program[5] we

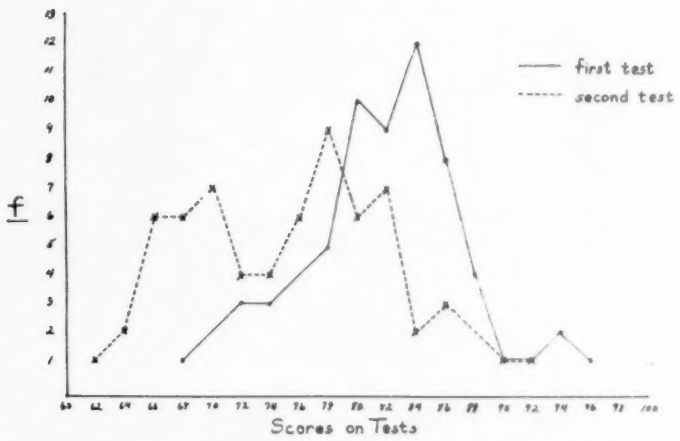

Fig. 3 — Distribution of scores of two identical rehabilitation examinations given senior medical students one year apart.

utilize four areas for such teaching: (1) the Respiratory and Rehabilitation Center at Creighton Memorial St. Joseph's Hospital, (2) the Department of Physical Medicine and Rehabilitation at the Veterans Administration Hospital, (3) the St. Vincent's Home for the Aged, and (4) the Outpatient Dispensary.

It has been our experience that on the general medical and surgical wards the teaching is more disease-centered and a large portion of the time must, of necessity, be directed towards the underlying pathologic and physiologic processes that are involved in the particular disease entity which is being treated, so that relatively little time can be allotted to the more comprehensive care aspects of the patient.

On the other hand, in the demonstration areas which I have mentioned, the turn-over of patients is relatively slower and a considerably greater portion of the entire staff's attention is patient-centered so that more teaching can be directed for the student's benefit. In our experience, this regularly results in carry-over to subsequent general medical and surgical wards experience for the students. If the student encounters rehabilitation experience only in the diluted experience of the general medical and surgical wards, the concepts will not have as much opportunity to compete with the older physiologic and pathologic approaches which every medical student should be required to master.

It is not intended that the teaching of the concept of rehabilitation should replace traditional approaches to medicine, but rather that it supplement such approaches. For example, if a student has a patient with senile osteoporosis with compression fracture, it would not be enough to teach the student the contribution that the social worker, psychologist, and physical therapist may make to the patient's rehabilitation. It is also necessary that the student know the basic physiology of calcium metabolism and the factors that may be etiologic in such a disorder. Rehabilitation medicine, therefore, is not a substitute for sound basic training in medicine. The former should presuppose the existence of the latter and then supplement it.

In our program we feel that those who perform rehabilitation services should be the ones who teach it to the students. Since rehabilitation services are provided by many different people, both medical and co-professional, naturally there will be many different individuals who will be teaching some segment of rehabilitation. A concept as broad as this scarcely can be presented in all its applied aspects to the student by one type of teacher. Many individuals among the co-professional groups can provide

demonstrations for the students that are very informative and, in addition, do much to enhance the students' appreciation of the worth of the co-professional groups. If the student fails to find in the faculty a sense of appreciation of the contributions which co-professional personnel can give the patient, he is not likely to utilize them following his graduation.

Ward rounds on someone else's patients do not provide the student the stimulus that comes from working with the disabled patient. Therefore, each student is required to work up at least seven to nine patients while in the demonstration areas. This makes his contacts with co-professional personnel more meaningful.

A service demonstration area provides the best opportunity for the medical student and co-professional personnel to measure each other, for in such an area he finds the medical faculty members appreciating most the contributions that other members of the team can make. A demonstration area usually has the 10 to 20 per cent of patients who require a team approach. This is valuable experience, but one disadvantage is that the student thus may feel frustrated since he knows he probably will practice where no team is available. It alone might leave the student with no taste for the concept of rehabilitation. It, therefore, must be supplemented by teaching in many situations where a team is not needed, such as outpatient departments of physical medicine and rehabilitation service units in general hospitals.

What Should Be Taught?

In various teaching conferences on rehabilitation, outlines of the basic technics which are considered important for the student usually are presented. I always have been impressed by the lack of agreement as to content. In our school we still adhere to the principle that since a sizeable number of our medical students will ultimately become general practitioners, and since many of them will intern in institutions which do not have adequate demonstration areas in the concepts of rehabilitation, it is necessary that the students understand basic principles such as prevention of deformity and decubiti, bowel and bladder rehabilitation, measurement of physical impairment and of disability, evaluation of activities of daily living, psycho-social adjustment, and pre-vocational exploration. They should know the underlying physical and physiologic principles upon which the use of physical agents is based. They should learn of the contributions electrodiagnosis can make in medicine and the part disability evaluation plays in patient diagnosis. They should be familiar with the fundamental principles involved in the rehabilitation of common disorders such as hemiplegia, heart disease, arthritis, amputations, traumatic disorders, and common neurologic disorders. They should also understand the essential contributions that are made by the physical therapist, the nurse, the occupational therapist, the psychologist, the vocational counselor, the medical social worker, the speech and hearing therapist, and the orthetist. The student should have contact with methods employed to utilize community and national resources for the disabled. He should know the manner in which native intelligence, interest, aptitudes and motivation influence the goals of the patient. The student should understand the variability of goals which may be attained by the individual patient; these goals include self-care, communication, ambulation, and work in either a homebound situation, sheltered workshop, or competitive industry.

It seems inconsistent in curriculum planning to require the student to spend a great deal of time learning about uncommon disorders such as diphtheria and typhoid fever, and then deny him adequate information in many of the chronic diseases which are continuing to arise in number and economic cost to our society.

It may be argued that much of this material should only come at the postgraduate level, but I would rather hold that early competition for the student's attention is needed. Studies[6] show an increasing cynicism in the medical stu-

dent as he moves toward graduation, and cynicism does not aid a concept of rehabilitation.

Another point concerning the teaching of rehabilitation should be made. If one is going to teach a concept adequately, it is important that it be introduced into the curriculum early, particularly in the pre-clinical years, and furthermore, that the actual experience concerning disabled patients be made early in the clinical curriculum and then later repeated. We have accomplished this by having conferences in rehabilitation for the freshman students. In the second year the measurement of disability is presented in physical diagnosis. In the third year we have continued our interdepartmental clerkship in rehabilitation and geriatrics, and finally, in the fourth year the principles of rehabilitation that the students have previously learned in their inpatient clerkship are applied in the outpatient clinics of arthritis, neurology, cardiology, pediatrics, orthopedics, otolaryngology, and psychiatry. In addition, in the rehabilitation clinic they have another opportunity to participate with the team of physicians and co-professional personnel in selected outpatients. Such an approach, we believe, fosters the continuing development of the concept.

Effectiveness of Teaching in Rehabilitation

When one measures the effectiveness of the teaching of anything, one must deal with two aspects. The first of these is content and the second is attitude. We have no objective information concerning either of these. At the end of the second year of our teaching program, we repeated with the senior class an objective examination that previously had been given them at the end of the clerkship in their junior year. While such an examination was fairly elemental and could be open to some criticism, it must be remembered that objective examinations in the area of rehabilitation are rather difficult to compile.

A comparison of the scores obtained after one year's interval indicated that there was a statistically significant amount of forgetting of subject material; however, even though students demonstrated a decrease of as much as 25 per cent and the over-all decrease was significant statistically, it was our opinion that the retention was better than we had expected. Since then, we have given many objective tests to our clerks at the end of their clerkship and have been satisfied with the amount of material which they retain as compared to their retention in other areas of medicine.

The measurement of attitudes, however, is much more difficult and we have no objective test which could give us information concerning this. This can only be measured by noting their increased concern about rehabilitation on the general medical and surgical wards and the outpatient department. Not all of them by any means are imbued with enthusiasm. In many instances, we regard it a gain to have given them understanding and passive acceptance of rehabilitation medicine. We believe that later experience in actual practice will confront them with recognition of the value of their particular training in rehabilitation.

Conclusions

Our experience over five years has convinced us that it is illogical to assume that exposure to concepts of rehabilitation will result in a whole-hearted acceptance of them by all students. Neither can all students be expected to accept psychiatric concepts or concepts of preventive medicine. Some students are by temperament unsuited to work with disabled patients, others by inclination are not interested in chronic disease which to them may appear both unprofitable and unrewarding in terms of end results. In each class, however, we have found a number of students who have exhibited much above average interest in the problem of chronic illness and the comprehensive care that is involved in such illness. We feel that this does not represent more than 10 to 15 per cent of the class. The enthusiasm of this group is reflected in their approach to patients when they return to the general medical and surgical wards, their manner of handling the patients they see in the outpatient

department, and the type of careers which they seek subsequent to graduation. This may appear to be a small number, yet if one considers that, as Shands[7] has stated in the past, there probably are not more than five per cent of the physicians in the United States who are enthusiastically interested in the problem of rehabilitation, increasing this group to 10 to 15 per cent in our schools of medicine will create a large enough nucleus of physicians for the future who can meet the challenges that lie ahead.

In closing, it is worth remembering, "you cannot teach a man anything, you can only help him find it within himself" — Galileo.

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It's no use growing older if you only learn
new ways of misbehaving yourself.
— SAKI

Mineral Metabolism Following Poliomyelitis

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● Controlled calcium and phosphorus balance studies have been performed in over 50 poliomyelitis patients whose degree and location of paralysis varied widely. During early convalescence, the intensity of hypercalciuria was nearly as great in mildly paralyzed patients as it was in the severely paralyzed and immobilized patients. However, the duration of hypercalciuria was directly related to the extent of paralysis: increased urinary calcium persisted in paraplegics for over six months and in many quadriplegics for over a year. Active and passive physical therapy, passive standing (tilt table) and early ambulation on crutches were evaluated for their effect on hypercalciuria. Physical therapy had no detectable effect on hypercalciuria and forced early ambulation was, at best, of equivocal metabolic value. In reversing the mineral loss, the muscular capacity for mobilization was apparently more important than was actual mobilization. The metabolic effects of the hormone, Nilevar, were studied in nine patients. Calcium excretion dropped to normal levels within three weeks, but a rebound of hypercalciuria followed drug withdrawal so that the net mineral loss was little changed by the therapy. Special attention was paid to preventing urinary calculi. Calcium intake was limited to 500 to 700 mg. daily. Prone positioning prevented genitourinary stasis of crystalline material. Daily urine output was maintained between 1,500 to 2,000 ml. When this program was initiated promptly after illness, only 5.5 per cent of respirator patients developed urinary stones. When the program was delayed for two months to three years, 26.5 per cent of respirator cases developed lithiasis.

When treating the immediate physical limitations produced by muscle paralysis, physicians may overlook the potential importance of more remote metabolic changes which the paralysis induces. This oversight can have serious consequences, since almost all patients having muscular weakness show concomitant abnormalities in calcium metabolism. The mineral changes ensue whether the primary cause of weakness is muscular or neural, and if the latter, whether at or above the anterior horn cell level. Subjects with only mild weakness have no more than an associated local, asymptomatic and mild osteoporosis. With more extensive paralysis, however, the complications may range from pathologic fractures to extensive soft tissue calcification (table 1) producing effects that prove more permanently disabling than the original neurologic or muscular disease. The severe complications are mostly preventable, but such prevention requires constant prophylaxis while treating the underlying neuromuscular illness.

Poliomyelitis serves as an excellent prototype for studying the metabolic effects of neuromuscular disease and for separating changes attributable to denervation paralysis from more general catabolic reactions. Since the degree of muscular immobilization in poliomyelitis varies widely between cases with different degrees of motor involvement, the extent of paralysis can be quantitated and correlated with the intensity of metabolic abnormality. Also, intact sensation protects most patients against both the skin infections and prolonged catheterization which plague many other paralytic states and indirectly cause additional metabolic abnormalities.

Table 1: Complications of Abnormal Calcium Metabolism in Paralytic Disease

Osteoporosis with Pathologic Fractures
Genitourinary Tract Calculi
Renal calyces
Bladder
Metastatic Soft Tissue Calcification
Renal tubules
Muscle
Around joints and traumatized areas
Diffuse calcinosis
Myositis ossificans
Hypercalcemia (pseudohyperparathyroidism)
Encephalopathy
Renal damage
Hypertension

This paper reviews several aspects of abnormal calcium metabolism in poliomyelitis, with particular reference to studies performed in this laboratory. Limited data is presented which indicates that the findings in poliomyelitis are relevant to other acute paralyzing illnesses as well. It is hoped that two principal points will be clear from these studies: (1) abnormal calcium metabolism develops in nearly all patients with acute poliomyelitis (and, by inference, other acute paralyzing illnesses), and not merely in those with severe paralysis and immobilization, and (2) the complications of abnormal calcium

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metabolism in paralyzing illnesses are largely preventable.

General Background

The Effect of Immobilization and Remobilization. Workers have been aware for many years that paralysis-producing neuromuscular diseases create skeletal demineralization. Allison and Brooks[1] demonstrated experimentally in 1920 that either immobilization or denervation produced local bone atrophy in dogs. During World War II, Freeman[2] and many others[3] recorded elevated urinary calcium levels in paraplegics. Howard and co-workers[4] consistently found hypercalciuria in patients with fractures and osteotomies; although bone trauma was directly involved, the immobilization of plaster casting appeared to contribute importantly to bone atrophy. Deitrick, Whedon and Shorr[5] definitively demonstrated in man that immobilization in plaster, without superimposed illness, denervation or muscle disease, was sufficient to induce moderately severe calcium losses. Somewhat later, Wyse and Pattee[6] quantitated the catabolic effects of paraplegia on calcium metabolism and demonstrated sustained hypercalciuria in every subject. In 1958, Whedon and Shorr[7] presented extensive observations on nine severely paralyzed patients with poliomyelitis. Every patient showed intense calcium losses lasting for an average of seven months.

The above studies leave no doubt that extensive paralysis or immobilization results in bone atrophy with consequent hypercalciuria. However, since nearly all the studies mentioned were performed on subjects with extensive physical limitations, they provide limited information on the range of calcium loss to be expected with paralysis of different degree or location, and might imply that demineralization is a phenomenon confined to the seriously paralyzed.

Although the metabolic effects of immobilization have received extensive study, only Wyse and Pattee,[6] Whedon and Shorr,[7] and Plum and Dunning[8] have presented adequate quantitative data on the metabolic effects of remobilizing paralyzed subjects. Several of Wyse and Pattee's patients already had urinary calculi, however, which made it more difficult to rely on the urinary calcium levels as reflecting body metabolism, and Whedon and Shorr's observations on mobilization were limited largely to the effects of the oscillating bed.

The Pertinent Physiology of Bone (fig. 1). Of the approximately 1200 gm. of calcium in healthy adult males, all but one per cent is skeletal.[9] If parathyroid and renal function is normal, serum calcium remains remarkably constant, despite prolonged or severe calcium deprivation. Serum phosphorus fluctuates more widely with dietary changes, but sustained alterations in serum phosphorus are similarly absent unless induced by humoral or renal disease.

Bone is metabolically active, undergoing continuous formation (through osteoblastic activity) and dissolution (through osteoclastic activity). Normally the formation and dissolution remain in close equilibrium, modified only by requirements for growth. Calcium-phosphate salts are deposited in an organic matrix (osteoid) producing a crystalline structure, similar to apatite, which composes the fundamental bone lattice. An additional 12 per cent of skeletal calcium is more readily mobilizable, being in a surface reaction with bone crystals and not bound by osteoid.[10]

Radioactive studies[11] indicate that about 20 per cent of the calcium and phosphorus ions of bone exchange fairly rapidly with extracellular fluids. When paralysis or immobilization disrupts the normal equilibrium, bone formation fails to keep pace with osteoclastic activity. Demineralization (osteoporosis) results, starting at bone surfaces.

The mechanism whereby paralysis induces bone resorption to exceed bone formation is unclear. There is little evidence that direct neural factors are responsible, since efferent nerves fail to terminate in bone.[12] Albright and Reifenstein[13] postulated that the tonic stresses and strains of muscular and postural activity are a necessary stimulus to maintain full osteoblastic activity.

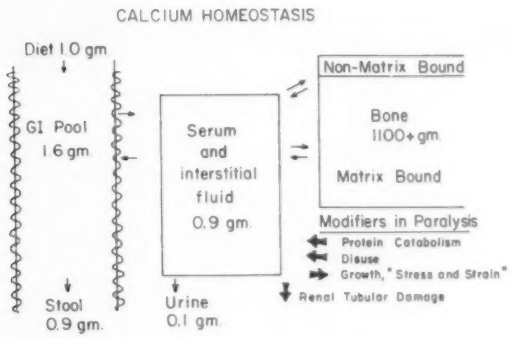

Fig. 1 — Calcium homeostasis in man. The regulating effects on serum calcium of the parathyroid glands are neglected in this diagram. Serum and interstitial calcium are in constant equilibrium with intestinal and bone reservoirs. Reducing intake has negligible effects on balance once growth has occurred. Osteoporosis shifts to the left the interstitial fluid-bone exchange, while renal tubular damage may deplete extraosseous reservoirs and increase the loss from bone.

Therefore, when motion and weight bearing are interrupted, bone matrix formation lags. The hypothesis explains many aspects of demineralization following paralysis but fails to explain the hypercalciuria observed in states like acute bulbar poliomyelitis where immobilization may be negligible.

Calcium Requirements and Excretion Pathways. Despite considerable variations in intake, the urinary calcium in health tends to be fairly constant and below maximal levels of 250 mg. per 24 hours in adult males and 160 mg. per 24 hours in adult females. (Actual values in most adults are generally considerably below these maxima.) In health, the daily adult calcium requirement is 0.45 to 0.55 gm. Children require more ingested calcium with levels being only approximately established and declining from 70 mg./K at three years to 12 mg./K at 16. Modest increases in calcium intake generally are reflected by an increased stool excretion, but more persistent and significant increases in intake (as in the milk-alkali syndrome) sometimes increase calcium absorption and, subsequently, urinary excretion. Although calcium is constantly exchanged between extracellular fluid and intestinal contents, little calcium is secreted into the intestine, so that increased mineral losses from bone principally act to increase urinary calcium content.

The renal threshold for calcium is a serum level of approximately 7 mg. per 100 cc. Above this level there is no necessary relationship between the serum calcium level (which reflects, among other things, parathormone activity and renal function) and urinary calcium. Thus, urinary calcium may reach twice the normal maximal levels when bone is being actively resorbed, while at the same time serum calcium usually remains normal or at the upper limits of normal. The factors which lead to increased renal excretion without an apparent increase in serum calcium levels are unclear. Occasionally, serum calcium does rise to abnormal levels in the presence of extremely rapid bone resorption (adolescent immobilization, extensive osseous metastases). Such hypercalcemia is always dangerous, for it indicates that the renal capacity for calcium excretion is exceeded, and that metastatic calcification and other metabolic problems are imminent.

The maximal daily renal capacity for calcium excretion is not clearly established. Examination of data from our own hypercalciuric subjects and from those reported in the literature reveals that when daily urine calcium excretion exceeds approximately 600 to 650 mg., serum calcium is usually elevated, suggesting that beyond this normal renal excretory mechanisms can no longer clear all calcium liberated from bone plus whatever is absorbed from the intestine.

Calcium Excretion in Poliomyelitis

Method of Studies. The data presented here were obtained employing conventional balance technics as previously described.[14] For the studies on the metabolic impact of mild bulbar poliomyelitis, nitrogen, phosphorus and calcium intakes were rigidly controlled as indicated on the balance diagrams. All stools and urine were collected in separate weekly pools and aliquots were analyzed for calcium, phosphorus and nitrogen using standard analytic methods. For most of the studies on urinary calcium excretion in poliomyelitis, calcium intake was limited to 500 mg. daily, and urine for analysis was collected in weekly pools for periods lasting several weeks or months. The constancy of creatinine excretion was utilized as an index to the accuracy of collections. Values are expressed as excretion per 24 hours. All patients received large fluid intakes (over 2000 cc.) throughout the study period. Patients with urinary tract calculi were omitted from study since the rate of calculus absorption or dissolution could not be controlled. The degree of paralysis and immobilization suffered by the patients is indicated in the appropriate text sections and diagrams.

Urinary Calcium Excretion During Convalescence. Twenty-seven patients with widely differing degrees of paralysis were studied to determine how the distribution and extent of weakness in poliomyelitis affected the intensity and duration of hypercalciuria.[14] The duration of individual study ranged from seven to 54 weeks with a mean of 20 weeks. Sufficient patients were studied early and late in convalescence to indicate reliably the duration of metabolic abnormality in each disability group.

Figure 2 lists the subjects according to extent of paralysis, age and sex, and roughly indicates the degree to which each was immobilized. Since patients within each disability grouping tended to show similar patterns of calcium excretion, the individual excretion data have been averaged together diagrammatically in figure 3. Urinary calcium excretion in figure 3 is expressed as a factor of the

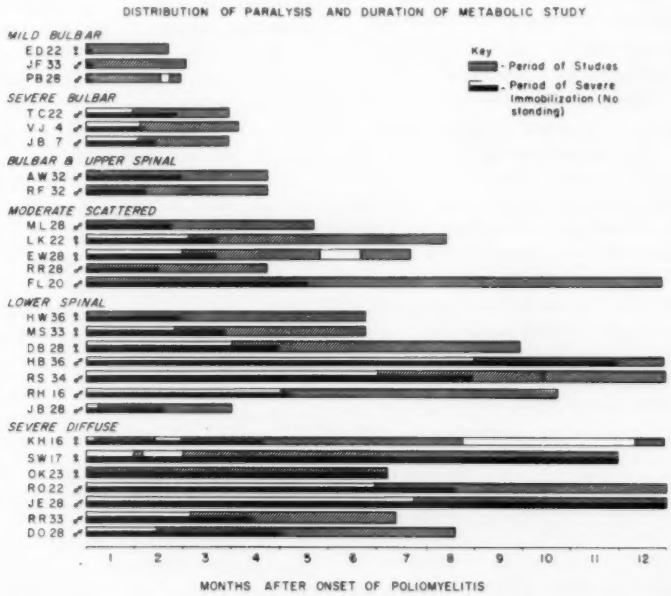

Fig. 2 — The grouping of individual patients according to extent of paralysis. Cross hatched areas indicate the periods during convalescence when metabolic studies were performed. Solid dark bars indicate the duration of bed rest prior to standing daily on a tilt table. In a few instances, patients were up in wheelchairs prior to standing.

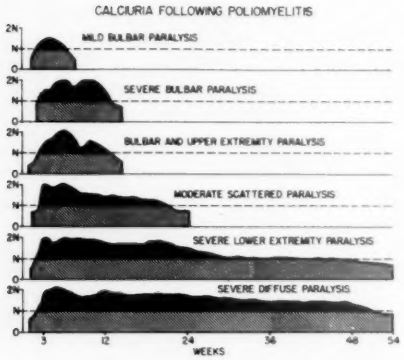

Fig. 3 — Urinary calcium excretion following poliomyelitis. The dashed line at N indicates the maximal predicted urinary calcium according to age and sex. Solid areas above N designate hyperexcretion. The excretion pattern in each disability group represents the average figure calculated from studies on all subjects presented in Figure 2. *(Reprinted from the A.M.A. Archives of Internal Medicine with permission of the publisher.)*

normal maximal 24 hour excretion level. This method of presentation was chosen since the study included men, women and children whose predicted normal maximal excretion varied widely. The specific individual excretion values can be found in a previous publication.[14]

As shown in figure 3, during early convalescence from poliomyelitis, urinary calcium excretion considerably exceeded normal in all groups studied. Calcium excretion rose above normal levels during the second or third week after the onset of poliomyelitis and then rapidly climbed to reach a peak approximating twice the normal maximum during the fifth to sixth convalescent week. This early hypercalciuria was significant in all groups and was nearly as intense in bulbar poliomyelitis patients with limited skeletal muscle paralysis as it was in extensively immobilized quadriplegics with almost no residual functioning extremity muscle. Such a similar level of hyperexcretion among the different groups was surprising, for if the calcium losses were simply the result of muscle paralysis producing immobilization, the degree of hypercalciuria should have paralleled the degree of paralysis.

Several possibilities might explain this relatively uniform early calcium loss, which correlated so poorly with the apparent degree of immobilization: one is that at twice the normal excretion level, calcium clearance by the kidney was at a maximum and further resorption from bone could not escape into the urine. This seemed unlikely, since serum calcium failed to rise in the more seriously paralyzed, and soft tissue calcification, which would have reflected diversion of calcium out of serum, also failed to develop. An alternative, and more acceptable hypothesis, is that the combined effects of acute illness and even limited paralysis induced maximal catabolism of bone, whose rate could not be accentuated even by more severe paralysis.

The Metabolic Impact of Mild Bulbar Poliomyelitis. Evidence that the systemic effects of poliomyelitis induced mineral catabolism independently of any immobilization is presented in figure 4 which diagrams the nitrogen and calcium balances of a young man with mild bulbar poliomyelitis who was not immobilized at all:

J. F., a 31-year-old school teacher, in September, 1955, developed malaise, mild headache, nausea and a temperature of 38 C. Within 24 hours he developed difficulty in, then paralysis of, swallowing. He was admitted to the hospital where right soft palatal, bilateral pharyngeal and left sternocleidomastoid weakness was noted. Spinal fluid showed 75 mg. of protein and 48 lymphocytes. By the day after admission he was nearly free of toxic symptoms and complained of hunger. A naso-gastric tube was passed and he was started on a stomach feeding. He was allowed to sit in a chair. By three days after admission he was free of fever. Detailed muscular examination showed the following weakness (1+ = minimal weakness, 4+ = complete paralysis): right orbicularis oculi 1+; right palatal musculature and right middle pharyngeal constrictor 4+; left palatal musculature and left middle pharyngeal constrictor 2+; left genioglossus 2+; left cleidomastoid 4+; right sternocleidomastoid 1+; right superior trapezius 1+; right triceps 1+; left abdominal oblique 1+.

He was allowed bathroom privileges on the fourth hospital day, walked one hour on the sixth day, and was placed on full ambulatory activity with active exercises on the seventh day. Thereafter, movement of his extremities was unlimited and he was transferred to the metabolic ward where he was completely self-sufficient and fully active, receiving occupational and physical therapy.

Pharyngeal function rapidly improved and the nasogastric tube was removed after the 17th hospital day. Thereafter he ate first a soft, then a regular diet, and his only reason for being in the hospital after the 18th day was for metabolic study which continued for a total of seven weeks.

Dietary calcium was limited to 200 mg. daily for the first 18 days, then increased to 450 mg. from the 18th to 24th day and 500 mg. daily thereafter. Nitrogen intake varied between 10 and 12 gm. daily, as shown in figure 4. Complete balance analyses could not be started until the 12th day because of loss of stool samples. Thereafter, he was in modest negative nitrogen balance for 18 days, following which he had a brief period of positive nitrogen balance, after which he stabilized. Calcium excretion exceeded calcium intake throughout the study period. Urinary calcium reached a peak of 370 mg. per 24 hours during the 24th to 30th day, and thereafter declined somewhat but did not fall below 250 mg. daily until the eighth week after onset of bulbar poliomyelitis (not shown on graph). At this time he had fully resumed his teaching duties. During the ninth week, a single 24 hour urine specimen showed 296 mg. of calcium. Further studies were impractical because of lack of dietary control.

This case illustrates how difficult it is to formulate in simple terms, such as "loss of stress" and "strain on bones," the mineral catabolic effects of poliomyelitis. While it might be inferred that calcium losses during the period of negative nitrogen balance reflected a catabolic failure to make osteoid, this hardly explains calcium losses exceeding negative nitrogen balance by several weeks. Dunning,[14] working in this laboratory analyzed urine 11-oxysteroids in a large group of poliomyelitis patients with hypercalciuria. Abnormal steroid excretion was absent, and variations in steroid output bore no relationship to calcium excretion. At present, therefore, the pathogenesis of the intense early mineral loss, which occurs so promptly in poliomyelitis and without apparent quantitative relationships to immobilizing paralysis, remains largely unexplained.

The Duration of Excess Calcium Loss. Once past the initial convalescent period during which all groups showed a nearly equivalent hypercalciuria, the pattern of

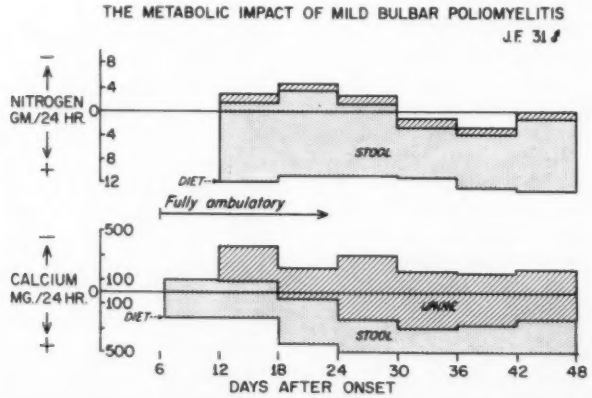

Fig. 4 — Calcium and nitrogen balance following mild bulbar poliomyelitis. Calculated nitrogen and calcium intakes are represented by the dark line at the base of each metabolic diagram. During periods when excretion rose above the zero line, the patient was in negative total balance.

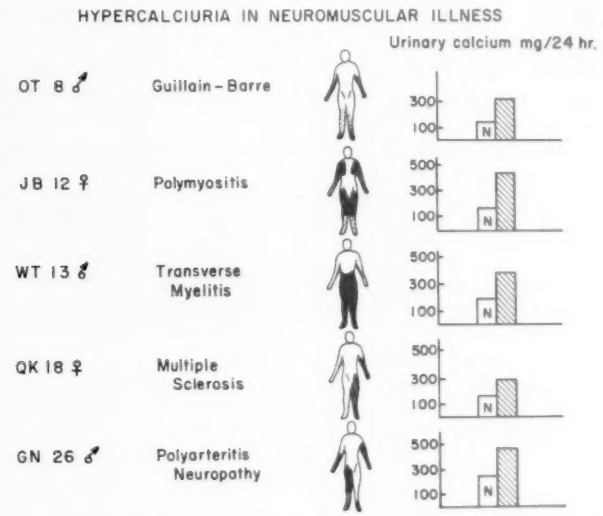

Fig. 5 — Hypercalciuria in miscellaneous neuromuscular illnesses. The extent of muscular weakness is indicated on the figures by shading: the darker the shading, the greater the weakness. Normal calcium excretion for age and sex is indicated on the clear, left-sided bar; actual excretion per 24 hours is cross hatched. All excretion values obtained between second and fifth month of illness.

mineral loss differed noticeably between groups and correlated better with the extent of paralysis (fig. 3). Thus, excessive urinary calcium levels were found in patients with bulbar poliomyelitis for only seven to 14 weeks, but progressively longer periods of high urinary calcium were found in each successively more seriously paralyzed patient group. Several quadriplegic patients, for example, continued to show elevated urinary calcium values for a year or more after the onset of poliomyelitis. Without total balance studies, it was not possible to estimate how much of the total body calcium stores were depleted in these subjects. Whedon and Shorr, who studied patients comparable to those included here with "moderate scattered" and "severe lower extremity" paralysis, estimated that their subjects had a mean calcium loss of 58 gm. This would amount to approximately five per cent of estimated body stores.

Relevance of Poliomyelitis Data to Other Neuromuscular Diseases. Mention has already been made of the hypercalciuria which others have demonstrated in paraplegia. Unfortunately, long-term studies comparable to those obtained in poliomyelitis do not exist in other neuromuscular diseases. Studies in this laboratory of daily or weekly calcium excretion in a variety of neuromuscular diseases (fig. 5) suggest that these other acute paralyzing illnesses produce abnormalities in calcium excretion similar to poliomyelitis.

Factors Enhancing the Development of Complications

Although every patient with poliomyelitis develops a negative calcium balance and high urinary calcium, only a minority suffer complications related to the metabolic abnormality. Data from other illnesses as well as poliomyelitis suggest several enhancing factors which increase the likelihood of complications. Some of these factors favor the development of several serious sequelae, others operate only to accentuate the hazard in a particular organ system. Since these factors may be at least as important as the underlying calcium defect, they are examined in greater detail.

Age. Osseus complications of abnormal calcium metabolism are more frequent in younger, growing subjects than in older patients. Bone atrophy so severe that

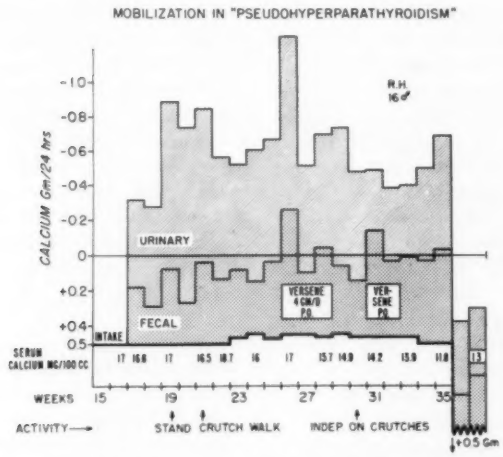

Fig. 6 — The effect of mobilization and oral chelating agents on hypercalcemia and hypercalciuria following poliomyelitis. Ambulation on crutches had little effect on serum or urinary calcium levels in this 16-year-old-boy, who had headaches, nausea and reduced urinary function when this study started, 15 weeks after onset of poliomyelitis. The chelating agent, Versene, had no great effect on either stool or urinary calcium excretion. During the final two weeks of study, increasing calcium intake to 1.5 gm. increased stool calcium moderately but also was followed by a rise in serum calcium to 13 mg./100 cc. ***(Reprinted from the A.M.A. Archives of Internal Medicine with permission of the publisher.)***

pathologic fractures were incurred during physical therapy were observed in only two of our subjects, both children under 10 years of age. Cases reported in the literature have been similarly youthful.

Adolescents may have particular problems consequent to sudden paralysis and immobilization. Even without particularly high calcium intakes, loss of calcium from bone in paralyzed or immobilized young people may be so rapid that renal excretion mechanisms are exceeded, leading to hypercalcemia and a clinical picture which Albright *et al*[15] have termed "pseudohyperparathyroidism." The hypercalcemia produces renal tubular damage,[16] with or without visible calcinosis, arterial hypertension, encephalopathy and, at times, soft tissue calcification. Similar syndromes are not seen in adults with post-paralytic hypercalciuria but may accompany Paget's disease or widespread osteolytic bone metastases as in myeloma or breast cancer.[17]

Rapidity of Immobilization. By and large, the soft tissue and renal complications of post-paralytic demineralization are confined to patients who have been suddenly immobilized following previous periods of relatively good health. Renal calculi, pseudohyperparathyroidism, and soft tissue calcification fail to complicate the osteoporosis which accompanies advanced muscular dystrophy. This appears to reflect the rate of calcium loss, dystrophy patients developing their advancing weakness so slowly that negative calcium balances are barely detectable. Thus, in dystrophy patients, pathologic fractures following falls or other gross trauma are essentially the only deleterious side reactions to bone atrophy.

Excess Calcium Intake and Dehydration. Intestinal calcium absorption is only partially regulated by bodily calcium demands. Although urinary calcium excretion may approach or exceed calcium intake in paralyzed or immobilized subjects, some ingested calcium often continues to be absorbed. Increasing the oral calcium intake in such patients increases somewhat the amount absorbed even when hypercalcemia has developed (see last two weeks of study depicted in figure 6). There is no evidence that osteoporosis can be minimized by increasing calcium intake, since the high urinary wastage reflects failure to utilize available mineral. Thus a high calcium

intake merely increases the amount of calcium salts presented to the kidneys for excretion, and if renal mechanisms already are functioning maximally, the calcium may be diverted into other non-osseus body tissues. One previously healthy 30-year-old woman, subsequently admitted to our care, developed diffuse renal calcinosis within seven weeks of contracting severely paralyzing poliomyelitis. She had been receiving an almost exclusively milk and ice cream diet, containing an estimated 1.8 gm. calcium daily. Her serum calcium was 11.9 mg./100 cc. and urinary calcium was 375 mg./24 hours at the time x-rays showed the renal calcification.

Hypercalciuria and hyperphosphaturia increase the amount of urinary solutes. Dehydration will increase this urinary solute concentration, and severe dehydration in normal persons is sufficient to result in solute precipitation in the urinary tract. Thus dehydration may be a factor in causing urinary calculi in patients with immobilizing paralysis. Whether renal calcium clearances can be increased by hydrating patients with hypercalcemia has not been carefully studied.

Steroids. Corticosteroids reduce bone matrix formation and induce osteoporosis and hypercalciuria.[17] Corticosteroids are rarely employed for most paralyzing illnesses, but are frequently used to treat patients with muscular weakness due to polymyositis, and sometimes used for patients with multiple sclerosis. We have observed pathologic spine fractures in two patients treated chronically with corticoids for non-immobilizing multiple sclerosis. In both instances, the spine fracture added more disability than any benefit gained from steroids. It is apparent that in a weakened or immobilized patient long-term corticosteroids are contraindicated except to treat specific lesions (e.g., muscle inflammation).

Muscle Trauma and Inflammation. Muscle calcification around large joints is a fairly frequent complication suffered by paraplegics and sometimes is seen in paralyzed limbs in poliomyelitics as well. Diffuse muscle calcinosis and even myositis ossificans may complicate polymyositis[18] but rarely has been reported in poliomyelitis.[19] The pathogenesis of these changes has not been worked out with certainty, but it appears likely that the abnormal calcium salt deposition is the result of connective tissue and muscle damage, possibly combined with slightly increased calcium phosphate levels in the interstitial fluids. It has been demonstrated that calcium phosphate is deposited in areas of muscle necrosis, even when serum calcium levels are normal.[20] Too vigorous physical therapy, producing tearing and small hemorrhages in periarticular connective tissue and muscle, combined with the mildly elevated serum calcium levels found in many paralyzed subjects, would appear to create optimal circumstances for pathologic calcification. In polymyositis, muscle inflammation is the fundamental pathologic alteration; it is hardly surprising, therefore, that calcific deposits so frequently are found in the muscles of subjects with this disease.

Urinary Tract Infection and Recumbency. These seemingly disparate factors are discussed together since urinary calculi are rare unless urinary infection is present. Even in infected subjects, calculi are most frequent in those who have been supine for long periods.

The importance of urinary infection in the pathogenesis of stones is evidenced by comparing the incidence of urinary calculi among paraplegics with that among poliomyelitics. In paraplegics who have lost bladder control and need chronic catheterization, as many as 50 per cent develop renal or bladder calculi.[21] By contrast, urinary calculi in poliomyelitis essentially are confined to patients who have been at least briefly catheterized and who have been recumbent (as in respirators) for many months.[22]

The normal urinary pH is acid and calcium phosphate is soluble in an acid medium. Since urea splitting organisms cause most urinary infections, infection produces an alkaline medium in which calcium phosphate precipitates. Infection-produced denudation of the urinary tract and exudates of organic material facilitate stone formation by providing a suitable

matrix. If the patient is kept recumbent, any urinary precipitate tends to drift into and remain stagnant in the dependent calyces. The combination of an increased calcium phosphate excretion, a urine pH favoring crystalline precipitation, a readily available organic detritus for matrix formation, and stagnant regions from which drainage is limited, creates a formidable risk of calculi.

Measures to Prevent or Minimize Complications

The measures which have been employed to try to prevent the complications of paralysis-produced bone atrophy fall under three general headings: (1) efforts to prevent or minimize the decalcification of bone; (2) efforts to reduce calcium absorption, or to draw calcium into the gut for excretion, and (3) general measures to avoid the risk of complications without directly trying to arrest bone atrophy or inhibit hypercalciuria. Of the three categories, the third is most tedious to achieve but carries the greatest likelihood of success.

Prevention or Minimization of Bone Atrophy. Two principal technics have been tried to reach this goal: artificial mobilization, using a variety of physical therapy technics, and anabolic steroids.

Several indications suggested that artificial mobilizing technics might ameliorate bone atrophy and mineral depletion in paralyzed subjects. Since immobilization was postulated to play a large role in creating demineralization, weight bearing and other physical activity to place stress on bone was a logical antidote. Whedon, Deitrick and Shorr's[28] early studies using the oscillating bed to treat young men immobilized in plaster appeared to support this presumption: during periods of oscillation, abnormal calcium excretion was reduced by half. Unfortunately, no such beneficial effects

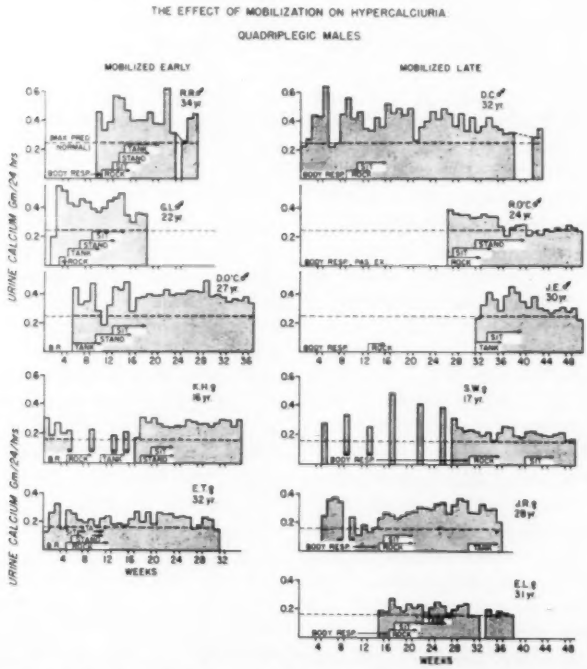

Fig. 7 — The effect of artificial mobilization on hypercalciuria. The patients in the left hand column were mobilized during early convalescence. Those in the right hand column were not activated until much later. The subjects were quadriplegic and comparably paralyzed. No apparent difference in excretion separates the two groups. Rock = patients using rapid rocking bed, usually 12+ hours daily. Tank = Hubbard Tank exercises. *(Reprinted from the A.M.A. Archives of Internal Medicine with permission of the publisher.)*

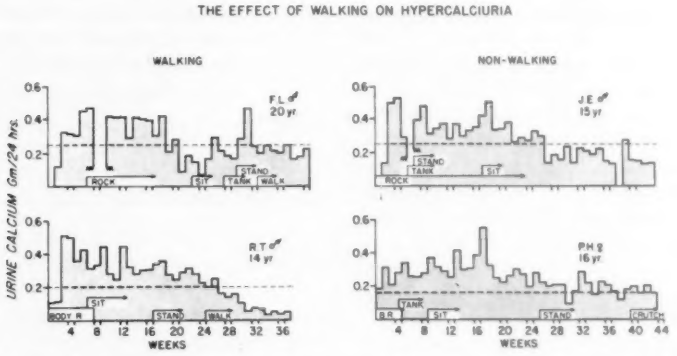

Fig. 8 — The relative effects on hypercalciuria of the muscular capacity for walking compared with actual walking itself. All patients had moderate scattered paralysis and were capable of walking by the 28th to 32nd week of convalescence. However, ambulation was delayed in the two on the right because of back problems. No apparent difference in calcium excretion separates the two groups. ***(Reprinted from the A.M.A. Archives of Internal Medicine with permission of the publisher.)***

have been observed when either the oscillating bed or other mobilizing procedures have been used on patients with muscle paralysis. Wyse and Pattee noted no change in calcium excretion when paraplegics were treated with the oscillating bed and passive standing. Whedon and Shorr similarly could detect no favorable influence on the negative calcium balance of poliomyelitics treated with the oscillating bed. Finally, Plum and Dunning[8] carefully monitored calcium excretion in a large group of paralyzed poliomyelitics who were treated with passive bed exercises, in the Hubbard tank, on a tilt table and even with crutch walking. No significant differences in excretion were noted coincidentally with initiating the mobilization, and the duration of hypercalciuria in rapidly mobilized patients differed little from the excretion pattern of other similarly paralyzed but less rapidly mobilized patients (fig. 7). By contrast, patients who regained sufficient muscle function to permit walking showed a concomitant cessation of hypercalciuria when walking became possible (fig. 8). Since the reduction of calcium loss was similar whether or not the patient actually did walk at the time, the muscular capacity for mobilization appeared to be more important in reversing demineralization than the mobilization itself. The results demonstrate that although passive standing and other physical therapeutic technics used to treat paralyzed patients may have merit for other purposes, none of the technics can be expected to halt the drainage of mineral salts from bone.

Once gonadal steroids and their synthetic congeners were demonstrated to affect favorably nitrogen and mineral metabolism in other osteoporotic states, it was natural that they should be tried in paralytic illness. Armstrong *et al*[24] demonstrated several years ago that these hormones reduced post-paralytic bone atrophy in experimental animals. Whedon and Shorr employed testosterone propionate and estradiol, given individually and together, to treat seven of their patients with extensive paralysis due to poliomyelitis. When testosterone alone or both hormones were given together, urinary calcium excretion and negative calcium balances were reduced by 0.1 to 0.3 gm. daily. The data was too limited to conclude whether the addition of estradiol added any clear advantages.

In this laboratory, norethandrolone (Nilevar), a synthetic anabolic hormone, was studied for its effects on the hypercalciuria of nine extensively paralyzed poliomyelitics.[25] With the relatively high dosage which was used, 1 mg./K daily, every patient reduced his urinary calcium excretion to within normal limits (fig. 9). Mean urinary calcium excretion during treatment was 45 per cent less than during pre-treatment periods. However, upon cessation of treatment, the excretion

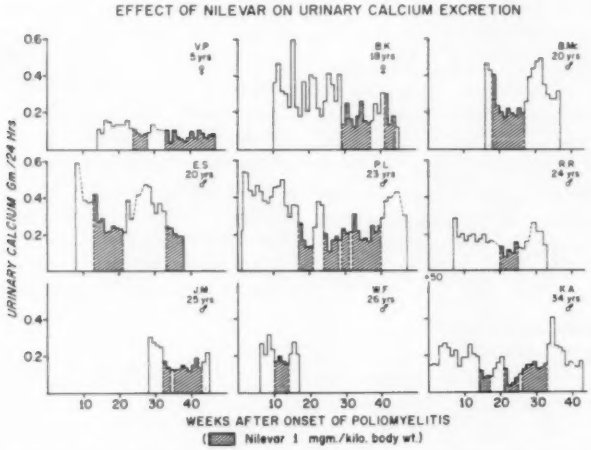

Fig. 9 — The effect of norethandrolone on hypercalciuria. The hormone was given in daily doses of 1 mg./K during the cross hatched periods. Following a brief lag period, calcium excretion in every subject fell to normal or near-normal levels. Discontinuation of norethandrolone was followed by a prompt rise in urinary calcium to reach or exceed pretreatment levels. *(Reprinted from the Journal of Clinical Endocrinology and Metabolism with permission of the publisher.)*

promptly rose again to pre-treatment levels or higher, suggesting a rebound loss of mineral. Two of the nine subjects developed mild cholestatic jaundice during treatment. One child, who was extensively paralyzed and not started on hormones until the 24th week of illness, developed pathologic fractures during the 32nd and 44th weeks, respectively, of her illness. During both these periods she was receiving the hormone.

It is difficult to be certain from either Whedon and Shorr's or our own study whether long-term use of anabolic hormones would materially reduce the degree of bone atrophy which ultimately follows paralysis. It is clear, however, that hypercalciuria can be at least temporarily interrupted, and this in itself can be valuable. Presently, we are using these drugs primarily to treat seriously immobilized and recumbent patients in whom urinary infection creates a high risk of calculi. Since the drugs are expensive and carry some risk of side reactions, they are discontinued once patients can be regularly gotten from bed or when urinary pH can be kept acid.

Reduction of Mineral Absorption. Urinary calcium hyperexcretion is little affected by reducing dietary intake when active bone resorption is taking place. Among our own patients, urinary calcium levels changed little during periods when patients were on calcium-free intravenous fluids. This could suggest that little value can be expected in the usual case by binding ingested calcium in the gut.

Two efforts have been reported to divert excess calcium losses from the urine to the stool for excretion. The chelating agent, sodium edathamil, binds calcium and is absorbed in negligible quantities when given orally. One of our patients with marked hypercalciuria and hypercalcemia, received edathamil, 4 gms. daily, for two separate periods lasting three and two weeks, respectively. Stool calcium increased only transiently, and urinary calcium excretion showed no appreciable decline (fig. 6).

Vagelos and Henneman[26] pursued a similar principle, using oral sodium phytate, which forms insoluble calcium salts, to try and reduce hypercalciuria in poliomyelitis patients. Urinary calcium was checked once a week and control values showed wide variations in most subjects. However, five of nine patients had reduced urinary calcium levels during phytate therapy. Unfortunately, stool calcium levels were not reported, so it is not possible to determine whether

calcium had actually been diverted to the intestine as was postulated.

General Measures. The above comments make it apparent that special treatments may temporarily ameliorate the rate of bone demineralization and urinary hypercalciuria following paralysis, but that effective prevention of the metabolic changes is presently lacking. This being true, major efforts are required to minimize the potential complications of calcium loss.

Some of these prophylactic measures are self-evident from the nature of the complications. It is doubtful whether pathologic fractures can be avoided completely, since prevention of demineralization is impossible and the physiotherapeutic procedures which produce the fracture are required to restore the movement which might re-mineralize bone. Pathologic soft tissue calcification around joints seems to require that the tissue first be damaged. This can only be avoided by therapists gently manipulating patients with limited range of motion. In polymyositis, it is conceivable that early and sustained suppression of muscle inflammation by steroids may minimize muscle calcinosis. This supposition needs clinical study. Adolescents with pseudohyperparathyroidism certainly appear to warrant treatment with testosterone proprionate, norethandrolone, or related compounds in an effort to reduce hypercalcemia. Although other reports in the literature suggest that early ambulation helps this adolescent response, our own patient showed no decline in his serum calcium (of 17 mg./100 cc.) for nine weeks after standing and six weeks after starting crutch walking (fig. 6).

The major danger of paralytic-induced osteoporosis is renal calculi. Fortunately, prophylaxis has great value in preventing this complication. The role played by hypercalciuria, infection and stasis in producing calculi has already been mentioned. Prophylaxis is simple, but requires constant attention by the staff. To prevent calculi, we have employed a program in which calcium intake is limited to 0.5 to 0.7 gm. daily. Fluids are forced to produce a measured urinary output of 1500 to 2000 ml. daily. Urinary tract infections are treated promptly with specific antibiotics (prophylactic or chronic antibiosis being of little value), and particular efforts are made to turn patients prone (even in respirators) early in convalescence. Of the various measures, insuring the high urinary output is the most difficult, but its importance justifies the constant attention which is required for success. The results of this program can be seen in table 2. The incidence of urinary calculi in respirator patients reported from other centers has ranged from 15 to 40 per cent. Similarly, of respirator patients treated by ourselves late in convalescence (without early prophylaxis), 27 per cent had calculi. By contrast, only six per cent of respirator patients receiving the above-outlined prophylaxis from the time of onset of poliomyelitis developed urinary tract stones. Comarr,[27] employing in paraplegics a regimen similar to that outlined above, has reduced the incidence of renal calculi from 12 per cent to 1 per cent. These results provide further evidence that painstaking preventive measures are effective in minimizing the complications of hypercalciuria.

Table 2: Incidence of Calculi in Long-Term Respirator Patients

Author	Total No. Patients	% Calculi
Taylor *et al*[28]	98	34
Slotkin *et al*[29]	47	40
Rodgers *et al*[30]	66	33
Affeldt[31]	675	15
NWRC* untreated	34	27
NWRC early treated	55	6

*Northwest Respirator Center of the University of Washington.

Summary

Osteoporosis with at least transient urinary calcium elevation is a constant sequel to paralytic poliomyelitis, whether the paralysis be mild or severe. During early convalescence, the hypercalciuria tends to be almost as great in mildly paralyzed as in severely paralyzed subjects. Later, the duration of calcium loss and

the consequent degree of osteoporosis bears a more direct relationship to the extent of paralysis.

Hypercalciuria is little ameliorated by artificial mobilization following poliomyelitis, the duration of calcium loss bearing a closer relationship to muscular return than to any specific activity which is instituted. Anabolic steroids will reduce calcium wastage, but because of their side reactions and expense are probably best limited to treating extensively paralyzed, recumbent subjects who are most threatened by urinary calculi or hypercalcemia.

To prevent the complications of abnormal calcium metabolism requires careful prophylaxis and constant vigilance. Urinary calculi are the most serious complications suffered by the seriously paralyzed, but can be almost completely avoided by attention to diet, position in bed, infection and fluid output.

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Moderation is a fatal thing.
Nothing succeeds like excess.
—OSCAR WILDE

Infirmary Rehabilitation of School Children with Cerebral Palsy

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● The authors have tried an infirmary rehabilitation of 10 children with cerebral palsy. The children were six to 15 years old. The rehabilitation treatment was divided into three periods: exercises in the hall, exercises in the water and pharmacologic treatment with Probamyl. All the children remained under a neurologic and electromyographic control—examination before and after each period of the treatment and at two months after the end of the treatment. The whole treatment lasted 10 to 12 months. A clinical improvement was found in all the children during the treatment as well as some time after its end. The clinical improvement concerned the improvement of the movements of the child as well as the diminishing of the spasticity. The rehabilitation treatment has in view the improving of the muscle coordination activity; on the other hand it influenced very little the strengthening of the myostatic reflex. It is possible in this way to create or improve the dynamic stereotype of movement. The effect of Probamyl, examined on the basis of electromyographic tracing, seems to strengthen the central inhibitory influences, which in effect gives the tracing a shorter time of action and lessens the range and extensivity of pathologic reactions. The analysis of clinical and electromyographic examination suggests that a joint pharmacologic and rehabilitating cure would produce a more advantageous movement result.

Ten cerebral palsied children, aged six to 15, normal or with slight mental deficiency, were received at a School Rehabilitation Infirmary, set up in connection with the Neurological University Clinic in Warsaw, Poland. An infirmary rehabilitation course was established to find out if the rehabilitation of cerebral palsied children could be undertaken with good results in an infirmary instead of in a rehabilitation sanatorium. If the infirmary treatment were a success it would reduce the number of patients in the sanatorium — where only children with severe motor disturbances would be directed — and it would permit leaving the cerebral palsied child with its family and among other normally, physically developed children. This latter point is confirmed by authors who also find it advantageous to leave the child at home.[1, 2] A change in the child's life influences its psychic equilibrium and lessens the possibility of rehabilitation. The example of normally developed playmates of the same age has a great importance in rehabilitation.

Five of the children under study were boys and five were girls. Six of the children were mentally at their chronological age; two were mentally retarded, and two were debilitated.

The children were given a neurologic examination which showed hemiparesis in five cases and paraparesis in two cases. Three cases showed spastic paresis of three limbs. Pyramidal syndrome with cerebellar ataxia was present in three cases. In one case there was hemiparesis with athetosis. In another case there was hemiparesis, athetosis, ataxia and intention tremor.

We chose Probamyl as the basic drug contributing to the rehabilitation as we consider that it fulfills all the conditions required from a drug used permanently by children with cerebral palsy. Probamyl reduces the muscle tonus;[3, 4] it does not lower the threshold of seizures and even in some cases removes or diminishes the amount of epileptic fits;[5-7] its action is prolonged; it has no influence on the vegetative system;[8, 9] it doesn't lower the scope of the child's attention and has a tranquilizing effect in some cases;[10, 11] it is not habit-forming;[12] it has no side effects; its metabolism occurs in the liver and only 12 to 20 per cent is excreted unchanged with the urine.[13]

The School Infirmary is of a general medical character so that the children had access to different kinds of specialists. They were under the permanent care of a neurologist, orthopedic surgeon, and psychologist, as well as an instructor of medical gymnastics. The children had permanent contact with the same medical rehabilitation staff.

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This contribution was read and approved by Dr. Irene Hausman, Director of the Neurological Clinic; Dr. Adam Gruca, Director of the Orthopedic Clinic; and Dr. Klemens Sokal, Director of the Department of Prophylaxis in the District Surgery of School Hygienics, Warsaw.

The rehabilitation included three different stages: gymnastics in the hall, gymnastics in the pool and pharmacologic treatment with Probamyl.

The division of the rehabilitation treatment was divided in three parts, in order to notice the clinical and electromyographic results of each separate stage of the treatment.

All the exercises were conducted individually because of the difference of age, mental development, different neurologic syndromes and range of disability of the children. The exercises always included general gymnastics, resistance exercises of the paralyzed muscle groups and correction of the child's posture and walk.

While directing gymnastics, the instructor became acquainted with the child; then he accordingly arranged further exercises and classified the children for exercises in the pool. Children with severe mental deficiency or frequent epileptic fits did not train in the pool.

Gymnastics in the pool were active and passive. Exercises lasted for three months and took place every other day. This kind of exercise was pleasant for the children and the parents so that the attendance increased.

After finishing exercises in the pool during the spring term the medical treatment began. Probamyl was administered after the rehabilitation treatment as a trial to maintain the achieved results of treatment. This exempted the child from attending the rehabilitation treatment during the busiest term at school. It also was easier to estimate the obtained results if the drug was administered without the coexisting gymnastics. Probamyl was administered in a daily dosage of 600 mg. for six to 12 weeks. This time of treatment depended only on the end of the exercises in the pool. For some children the period of exercises in the pool lasted longer because of intervals caused by sickness.

The orthopedic and psychologic examination took place before and after the treatment. The neurologic examination was given after each stage of rehabilitation as well as two to three months after the end of the treatment. The electromyographic examination lasted for a whole year; the children were examined before beginning the treatment, the first examination took place after about two months of exercises, the second after about three months of exercises in the pool, and the third in the third or fourth week of administering Probamyl. Further examinations took place between the fourth week or fourth month after administering Probamyl. The electromyographic examinations were done with the Schwarzer 8 canals apparatus, which makes it possible to compare the antagonists muscle groups of the thigh and crus of both lower limbs while in motion. Bipolar surface electrodes with a diameter 1.5 cm. were used. Needle electrodes were not used as this required repetition, to which the parents would not easily agree. For our purpose such potential records were sufficient to compare registration obtained during different periods in similar technical conditions. The muscles, tibialis anterior, gastrocnemius, rectus femoris and biceps femoris of both limbs were examined. The distance of the electrodes was 1 cm., calibration 160 microvolts according to the amplitude 10 mm. Resting potential in the recumbent position, after the child calmed down, were registered during movements of the foot, during the provocation of Babinski reflex, during the passive and sudden stretching and shortening of the muscles of the crus, during tendon reflexes and clonus of the foot and later during walking and standing.

The estimation of the treatment results was based on clinical and electromyographic examinations. The clinical improvement was stated during the treatment and directly after its end in all our cases. The most improvement shown was in the strength of the normalization of the muscle tone. In certain cases equalization of physiologic reflexes and lessening of spastic signs were observed. The symptoms in two of the four cases of ataxy disappeared completely and in two the ataxy significantly diminished. In two cases of athetosis the symptoms disappeared completely in one and in the other only its traces remained during the pharmacologic treatment.

The clinical improvement was spectacular in some cases as with a six-year-old girl with spastic paresis and marked ataxy of inferior extremities. While beginning the treatment the girl could not walk without help; she used to fall while walking, and was brought to the majority of exercises by her mother in a taxi. After finishing the treatment the girl walks easily without help; this improvement has lasted about one year.

In an eight-year-old boy with left-sided hemiparesis and cerebellar syndrome, the neurologic symptoms disappeared completely during the treatment and the child's movements were quite normal. In this case, however, the improvement has diminished with time.

In the case of a 12-year-old girl, with a slight hemiparesis of the left side, the improvement of the muscle strength was so great that the girl now trains for fencing competition from a reverse position.

The clinical improvement persisted in six cases for six months after the end of the treatment. In the four other cases a change for the worse was noticed during this period. In one case this change for the worse happened immediately after the administration of Probamyl was stopped.

Caution must be used in the comparative analysis of the results of exercises in the hall, exercises in the pool, and the pharmacologic treatment.

The clinical neurologic examination did not show distinct differences in the majority of the cases in the separate periods of the treatment but we gathered additional information from the children's mothers. Only in three cases was there an improvement in the strength of the muscle and a normalization of walk during the exercises in the hall. After the exercises in the water, an improvement was noticed in all the children, who became stronger and more efficient. The greatest changes were noticed during the period of Probamyl administration. In all the cases the movements of the affected limbs became easier and more confident. The children acquired new movements — for the first time they began to walk, to run and to go unaided down the stairs. One patient, who had always lost to other children began, for the first time, to win over his playmates at school. Great psychic animation was observed in single cases. It is interesting that the similar improvement was noticed in two children who received only pharmacologic treatment.

Observations based on the electromyographic examinations are as follows:

1. An electric (spontaneous) activity of the muscles noticed in three cases, among them one case of athetosis, diminished considerably in two cases after exercises in the hall and in the pool. It disappeared in all the cases after pharmacologic treatment. A trace of electrical activity appeared in two cases three months after stopping the pharmacologic treatment (table 1).

2. While examining the Babinski reflex we noticed a strong reaction of the tibialis anterior muscle as well as a reaction of the thigh muscles, rectus and biceps femoris, and of the symmetric muscles of the opposite limb in cases of hemiparesis or paresis of any limb. After exercises in the hall, and in the pool in six cases the reaction of the muscles decreased in different degrees. During pharmacologic treatment in two cases a slight reaction of short duration was noticed in the muscle of the extensor of the foot, and a disappearance of muscle reaction was noticed on the opposite side. In the remaining cases the difference was smaller. The effect of the treatment lasted for two months. After three months a trace of the increase of reaction was noticed in two cases, and after four months in two cases a distinct increase of the reaction in comparison to the period of pharmacologic treatment was found.

3. The passive reaction to stretching and shortening of the muscles disappeared completely during pharmacologic treatment in one case, and in two cases only its traces remain. In the other cases it remained in a much slighter form (table 2).

4. Before the treatment in two cases, while the foot was executing active movements, a higher amplitude of the antagonist muscles not allowing for

efficient movements, was noticed. In the remaining cases there was a lesser reaction of the antagonist muscles. The rehabilitation treatment of the two cases achieved a complete normalization of the antagonist muscles in relation to their antagonists. In the other cases there was a great improvement. In one case we did not notice any difference between the state of the patient before and after the treatment.

During and after the pharmacologic treatment a further increase of the antagonist muscles potential and a decrease of the antagonists was noticed in four cases; in four cases there was only a small difference, in two no difference at all.

5. In six cases before the treatment we noticed a disturbance in the organization of muscle activity consisting in an uninterrupted electric activity of the examined lower limb muscles as the patients walked. As a result of rehabilitation and pharmacologic treatment in five cases there appeared a differential activity potential, i.e. intervals in the working of the antagonist muscles during the contraction of their antagonists. The complete lack of improvement was noticed in one case only (table 3).

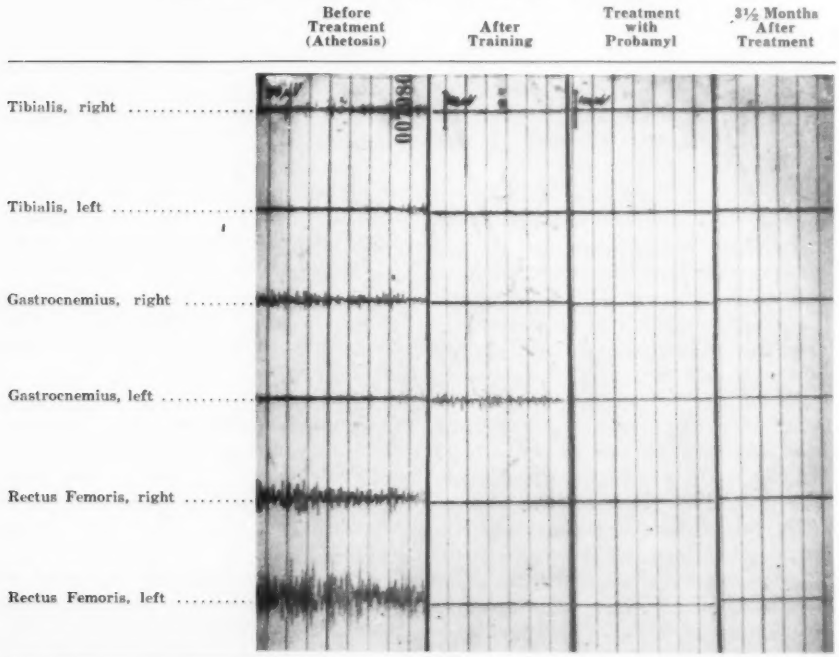
6. The permanent activity of the muscles in a standing posture, noticed before the treatment, decreased in three cases after the rehabilitation treatment and in the remaining cases after pharmacologic administration.

While comparing the electromyographic examination with clinical results one comes to the conclusion that the improvement in the picture of the electromyogram was simultaneous with the clinical improvement. In two cases a quantitative deviation was found. In one case the electromyographic changes are stated three months before appearing as a clinical change for the worse. In two cases of clinical changes for the worse this order of symptoms of the electromyogram was not noticed.

Case History

K. T was a seventh month baby, born in 1951 as a second and only living child

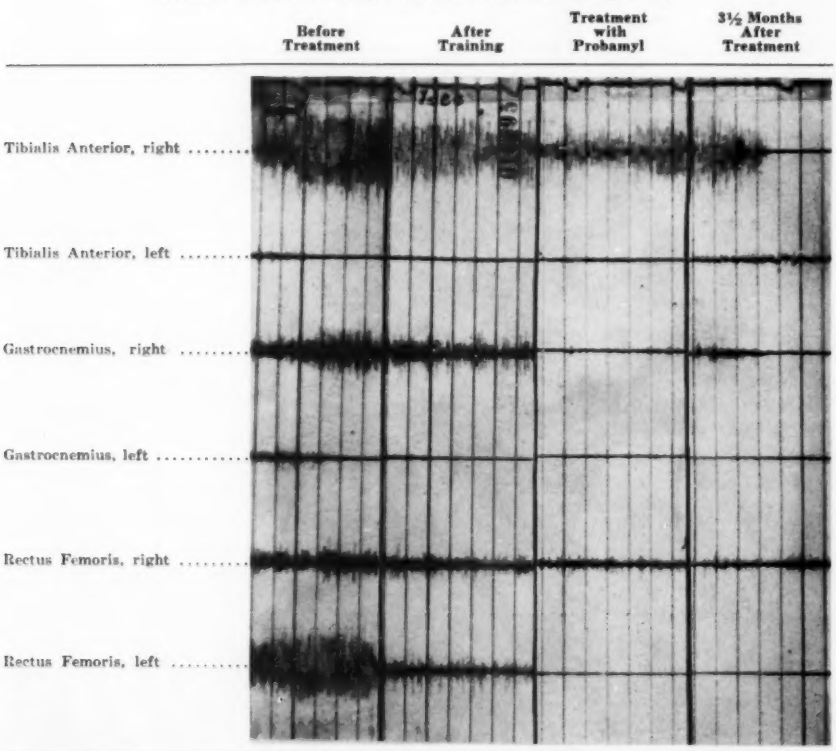
Table 1: Muscles Activity at Rest (Pyramido-Extrapyramidalis Syndrome)



from her mother's seven pregnancies. In the first day of her life a paroxysmal cyanosis was observed. She had a cyst on her neck. She did not react to visual impulses until her sixth month. In her fifth year, it was found she had bilateral atrophy of the optic nerve with great impairment of sight in the right eye and a slighter impairment in the left eye. She had tried to walk when one year old but had fallen constantly. She often had been examined by an orthopedist who didn't think she would be able to walk. Specialists of the children diseases suspected at first the consequence of infantile paralysis, later they diagnosed Little's disease. The girl's general physical development progressed normally but until her seventh year she only walked at home and with the help of another person. Her mental development always was considered normal. She had no contagious diseases except measles.

In October, 1957, an orthopedist directed her to a neurologist for rehabilitation treatment. The neurologist's observations included these findings: the child entered the doctor's room supported under an arm by her mother. Left to herself she became unsteady and fell. There was a slight ptosis on the left; the eye-balls were asymmetric; the left eye-ball receded upwards but did not reach the outer corner. There was right central facial nerve paresis. While at rest both feet turned inwards, the left foot more. The left thigh was thinner about one-half cm. than the right one, and the crus about two and one-half cm. thinner. The left leg was weaker than the right especially in the distal muscle groups. The muscle tonus in both legs had a flaccid character. Knee reflexes were weak on both sides. Achilles reflex on the right, plus on the left, paroxysmal contraction were observed of Achilles

Table 2: Stretch Reflex of Right Foot (Passive Flexion of the Right Foot)



tendon, so that no response could be obtained. Spastic reflexes were present bilaterally. There was ataxy of both sides. The girl stood with toes of both legs tucked under. Her gait was spastic with ataxy and tendency to fall.

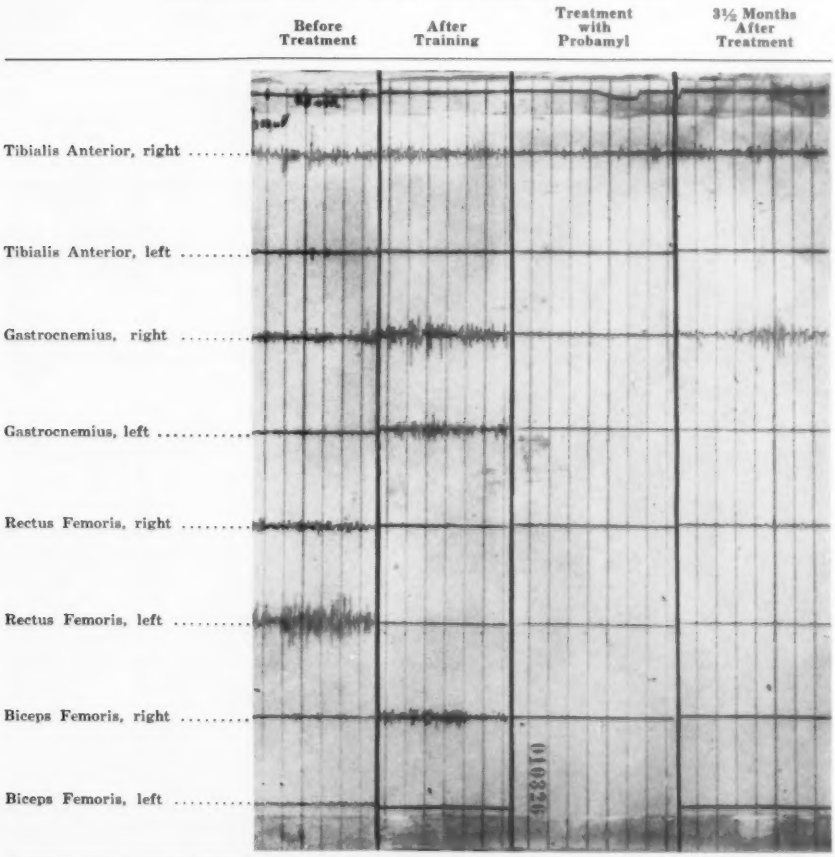
A psychologic examination showed the girl's mental development was adequate to her age. The girl was lively, excitable and reacted quickly. The contact was good. She was not self-dependent because of her illness.

The diagnosis: Little's disease.

The girl went through a full course of individual exercises in the hall and in the water with a special stress on walking, and pharmacologic treatment for six weeks with Probamyl. The daily dosage was 600 mg. During the exercises in the hall the girl began to walk without help and at the end of the exercises in the water she walked much better than at their beginning. She became more lively. The greatest changes noted during the treatment with Probamyl: the child became psychically more vivacious; her appetite improved, she began to run, to go upstairs by herself and to become quite self-dependent. In the first week of Probamyl treatment the contraction of left Achilles tendon disappeared, so that while walking the child placed her weight on the whole foot. She did not fall any more while walking or running.

The most important changes found by this neurologic examination compared to the initial one were: on June 11, 1958, after finishing the administration

Table 3: Static Electrical Activity (Pyramido-Extrapyramidalis Syndrome)



of Probamyl, the child entered the room without help and looked at ease in the consulting room. She stood normally on her feet; she walked placing both feet normally. Only when she was excited did she sometimes stand on the toes of her left foot. Her spastic gait was less pronounced but ataxy was evident as in the past. The Achilles reflexes were equally easy to provoke, Babinski's and Chaddock's sign on the left, on the right no spastic reflexes.

Examinations four months after the end of treatment showed the left foot was slightly weaker although the strength of the remaining muscle groups was good. The muscle tonus was normal. Knee and Achilles reflexes were equal on both sides; they were not exaggerated; Babinski's sign was positive on the left; Chaddock's sign was positive on both sides. Ataxy was visible only in the left extremity. Her gait was the same as in the previous examination. A psychologic examination after one year showed the child was quite self-dependent. She now attends a normal school and takes part in social life.

Electromyographic Examination of the Girl. Before the treatment began a rich electric (spontaneous) activity of the majority of examined muscles was observed. During the treatment with Probamyl the electric activity completely disappeared. Three months later — after stopping the treatment — a trace of electric activity in the left tibialis anterior muscle was present. The influence of rehabilitation on the electric activity was minimal.

While examining the Babinski reflex we noticed electric potential in all the examined muscles of both extremities, the highest in extensor muscles; this was greatly reduced during administration of Probamyl and there was a considerable lessening of the potential amplitude of the examined muscles. The influence of exercises on the extensity of Babinski's reflex was not marked. Three months after the end of the treatment the effect diminished but was still remarkable in comparison to the initial examinations.

During work: Before the treatment marked reaction of the antagonist muscles were obtained. During the pharmacologic treatment the antagonistic muscle reaction decreased two to three times. The effect of pharmacologic treatment was present three months after stopping the treatment.

During passive movements: A strong reaction of the tibialis anterior muscle to slow and sudden stretching and a permanent activity of the rectus femoris muscle was observed. We did not notice changes after the exercises. During the Probamyl treatment the reaction upon stretching of the tibialis anterior muscle was almost one-half lower. After three and a half months there was almost no muscle reaction to slow stretching and the reaction to sudden stretching was similar to the one during the treatment.

In a standing position: High activity of all examined muscles was observed. After exercises in the hall, the activity slightly increased, but after exercises in the pool it decreased significantly. During the pharmacologic treatment there was a slight activity of the right gastrocnemius muscle. After three and a half months there was a higher muscle activity.

While walking: There was a permanent activity of the left rectus femoris muscle and the other muscles showed a partly differentiated activity. During the pharmacologic treatment a considerable increase of the differential activity of all muscles was noticed. After a three and a half months interval in the treatment the effect of the treatment was only partial.

Conclusion: During the treatment spontaneous activity of the muscles was discontinued and there was a considerable lessening of the pathologic reaction of muscles on stretching during the Babinski reflex and while standing. There also was a differential activity of the muscles while walking. After a three and a half months interval the spasticity partly increased. In comparison with results obtained in other cases, this one must be considered as mediocre.

Conclusions

1. The rehabilitation treatment of children with cerebral palsy can be

undertaken with good results in an infirmary with lesser cost than in a sanatorium. This also allows leaving the children with their parents.

2. The rehabilitation treatment influences the coordination of the muscle work although, on the other hand, it influences only slightly the increased myostatic reflex. In this way it is possible to improve or produce the dynamic stereotype of the movement.

3. The effect of Probamyl, analyzed chiefly on the basis of the electromyographic tracing, seems to depend on the strengthening of the central inhibitory influences, which gives the tracing a shorter time of action and lessens the range and extensivity of pathologic reactions.

4. The analysis of clinical and electromyographic examinations suggests that the pharmacologic treatment with the rehabilitation would produce a better result of the motoric development of the child.

Summary

The authors have tried an infirmary rehabilitation of 10 children with cerebral palsy. The children were six to 15 years old. The rehabilitation treatment was divided into three periods: exercises in the hall, exercises in the water and pharmacologic treatment with Probamyl. All the children remained under a neurologic and electromyographic control-examination before and after each period of the treatment and at two months after the end of the treatment. The whole treatment lasted 10 to 12 months. A clinical improvement was found in all the children during the treatment as well as some time after its end. The clinical improvement concerned the improvement of the movements of the child as well as the diminishing of the spasticity. The rehabilitation treatment has in view the improving of the muscle coordination activity; on the other hand it influenced very little the strengthening of the myostatic reflex. It is possible in this way to create or improve the dynamic stereotype of movement. The effect of Probamyl, examined on the basis of electromyographic tracing, seems to strengthen the central inhibitory influences, which in effect gives the tracing a shorter time of action and lessens the range and extensivity of pathologic reactions. The analysis of clinical and electromyographic examination suggests that a joint pharmacologic and rehabilitating cure would produce a more advantageous movement result.

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Teaching of Rehabilitation in a Medical School

Report of Subcommittee
Committee on Professional Education
American Rehabilitation Foundation

One of the major road blocks in providing adequate rehabilitation services throughout the nation is the lack of training programs. Adequate teaching programs should provide increasing numbers of trained personnel to establish, expand and improve services in other universities and communities. Basically two types of teaching programs can be developed. One type is restricted to one phase of the training in rehabilitation, such as the training of undergraduate medical students. The effectiveness of such a program is limited. The second type is a comprehensive program which includes the teaching in a medical school, of all phases of rehabilitation which relate to physical disability resulting from neurological or musculoskeletal disorders, and rehabilitation of the mentally ill, the blind, deaf, and others. In addition, the structure of this type of program lends itself quite readily to the teaching of the multiple disciplines needed for rehabilitation in a common facility. If properly coordinated, the needed medical guidance is readily available, and each phase of the program lends strength to the other. Through the utilization of a common teaching staff and facility, a comprehensive teaching program is not only more effective but also more economical than numerous limited and isolated curricula. For all reasons, an effort is made to describe the standards of rehabilitation teaching programs within a medical school which would insure the highest caliber of training with maximal efficiency. It is essential to realize that the financial needs of such major programs must be viewed realistically, and monetary support be appropriately scaled to meet their cost. The development of new programs of this type is time consuming and occurs at a gradually increasing rate. With this in mind, much emphasis should be placed on expanding the scope of the program within a reasonably short period of time to meet the described criteria of a comprehensive program. In this way the teaching program will rapidly approach a high level of efficiency. In order to establish such comprehensive programs, grant support will usually need to extend over periods longer than three years.

Teaching Objectives

The following objectives are considered minimal in connection with training of medical students in rehabilitation. The clinical program should prepare the student to function on the level that is needed in general practice of medicine. Clinical instructions should be given in the diagnostic and treatment procedures in the field of physical medicine and rehabilitation as they can be used in general practice. At the same time, the student should learn how to evaluate properly patients with severe physical disabilities resulting from accident or disease. The student should become familiar with rehabilitation problems to such an extent that he will be able to recognize when the patient needs specialty care or special evaluation procedures. He should be taught to provide the necessary services for his patient by proper referral and by proper utilization of paramedical specialties, available community resources, and state-wide services. The medical student should learn to recognize that the physician's responsibility does not end with the treatment of an acute illness or injury. He should learn the importance of a positive but realistic attitude on the part of the physician toward the severely handicapped patient and the significance of the resulting patient-physician relationship to the success of the individual's rehabilitation program. He should be aware of the need for effective communi-

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cation and cooperation among the various professions concerned with the chronically sick and disabled, and the obligation of the physician to provide guidance to paramedical specialties.

Teaching Program to Undergraduate Medical Students

It is recognized that in order to fulfill the outlined objectives it will be necessary to establish the curriculum in physical medicine and rehabilitation as an integral part of the four-year medical school curriculum. It therefore will be necessary to modify the teaching approach according to the needs of each individual school. However, some basic concepts will be essential to any program which aspires to provide high standards of teaching in rehabilitation. It is most desirable that the students be oriented early in their medical career to recognize problems of the chronically disabled patient including social, psychological and vocational, as well as physical factors.

The following review is presented to illustrate these essentials. The medical school curriculum should contain instruction in functional anatomy from the standpoint of fostering understanding of the biomechanics of the musculoskeletal function. Basic biophysics should be taught as related to the therapeutic application of physical agents and procedures and as required for the understanding of other rehabilitation technics. These subjects should be introduced early in the curriculum, preferably during the first and second year.

Training in physical diagnosis should include the teaching of principles of kinesiology as related to physical diagnosis and evaluation of disability. This should include the teaching of tests and technics useful in the evaluation of joints, muscular weakness, body mechanics, and gait. This phase of the teaching program should be considered as part of the general instructions in physical diagnosis given in the beginning of the clinical years.

In the clinical years, the curriculum should emphasize the total evaluation of the patient including the evaluation of his disability, the assessment of his personality resources, the social factors involved, and his vocational potentials. Such an evaluation is essential as a prerequisite for outlining a treatment program. The student should learn how to outline such a treatment program in cases which can be treated in general practice. This will include the prescription of simple physical therapy and occupational therapy technics as well as other basic physical medicine procedures. The basic principles underlying the indications for simple types of orthetic and prosthetic devices should also be part of the curriculum. The student should become acquainted with the elementary training in the use of these devices.

There should be frequent presentations to emphasize the need for cooperation and comprehensive management of long-term illness. A working knowledge of contributions of the nonmedical specialties to the rehabilitation of the patient should be obtained by the student.

A research program should be established as an integral part of the teaching program. It is important to the teaching for several reasons. It is necessary for increasing the fund of knowledge in the field. In addition, faculty and student participation is a constant stimulus to enrich the teaching program through the evaluation of the latest developments.

Student participation in research encourages independent thinking and fosters a more critical attitude in his evaluation of research as published in the literature. It also serves to increase his awareness and understanding of the contributions of the specialty and of the problems involved in extending knowledge in medicine.

Residency Program

Staff and facilities available for the outlined undergraduate program in the medical school can also serve a medical residency training program in physical medicine and rehabilitation. Thus the greatly needed medical specialists in rehabilitation can be trained with little additional expense. At the same time the residents will contribute substantially to the undergraduate teaching program.

The presence of this residency program in physical medicine and rehabilitation

will contribute to the effectiveness of the graduate training programs in other medical specialties such as neurology; rheumatology; neurosurgery; orthopedics; internal medicine, and pediatrics.

Training of Occupational and Physical Therapists

The staff and the facilities of the medical school would also be available for training of physical and occupational therapists in their corresponding curricula. This would provide for the needed medical guidance in these areas. It would give the students enrolled in these curricula a much stronger experience in rehabilitation than they could receive in a limited isolated program oriented toward only one of these health professions. The therapy student would obtain a working knowledge of allied specialties working in rehabilitation and thus be prepared to work in a team situation where the problems of the patient require comprehensive rehabilitation.

At the same time the teaching faculty in the curricula in occupational and physical therapy would be available for teaching in the medical school graduate and undergraduate programs and thus contribute to the scope of these curricula.

Training of Other Paramedical Groups

A conjoint teaching effort as that described for occupational and physical therapy can also be used effectively for the training of speech therapists and speech pathologists. The faculty of the described comprehensive program could participate and contribute to teaching programs for rehabilitation counselors, medical social workers, psychologists, as well as the teaching of rehabilitation nursing in the school of nursing. For this latter purpose, a special program in rehabilitation nursing could be included. Since the students in the various specialties need to acquire a concept of comprehensive rehabilitation as well as obtain a working knowledge of the role of other professions and their contribution to the rehabilitation of the patients, such a joint program would be most effective. The joint utilization of staff and facilities for all these programs would allow an institution to gain the maximal benefit in teaching of rehabilitation in the most economical way.

In order to provide for a teaching program of this scope, it is essential to recognize the need of the following basic prerequisites in order to establish and maintain a high level of standards throughout all parts of the program.

The following will be necessary:

1. A full time academic and clinical faculty adequate as to quality and numbers.
2. Clinical services supporting the teaching program being jointly utilized by the various phases of the program.
3. An adequate teaching facility jointly utilized by all parts of the program.
4. An administrative plan of organization which allows rendering patient services and teaching to the various aspects of the program efficiently at a high level of performance.

Teaching Faculty

The director of such a training program at a medical school should be highly qualified by training and experience not only to organize and direct the training program but also to provide the clinical services essential for a good teaching approach. He should be a physician with a deep interest and demonstrated ability in teaching. He should have broad training in the clinical aspects of the musculoskeletal disabilities and in all diagnostic and evaluation procedures essential for successful rehabilitation. This includes experience with such new diagnostic methods as electrical testing and electromyography. He should have knowledge and experience in using the technics of physical and occupational therapy. He should have training, experience, and familiarity with the use of psychology, speech therapy, the social aspects of the disability, vocational counseling and the use of community resources as they are related to the disabled. He, therefore, should be a diplomate of the American Board of Physical Medicine and Rehabilitation. In addition, he should be assisted in his teaching efforts by an adequate number of board eligible physiatrists.

It is essential that a full complement of faculty in other medical specialties be available within the framework of the medical school.

It also seems to be essential for an adequate teaching program in a medical school to have available a paramedical teaching faculty with a special interest and training in rehabilitation. For example, the faculty member in psychology should not only be an expert in his own field, but have special experience with the problems related to physical disability. The availability of teaching faculty in physical therapy, occupational therapy, speech therapy, medical social work, rehabilitation nursing and rehabilitation counseling is also highly desirable for the reasons mentioned. Conjoint teaching of both graduate and undergraduate medical students, as well as the students in the paramedical specialties will enrich the experience of students in all the fields.

Clinical Services

From the students' point of view the clinical services can be looked upon as an extended laboratory experience. As in all health sciences, the teaching of rehabilitation is done to a large extent by example. Therefore, it is a prerequisite for any good teaching program that adequate clinical services are available for the teaching purposes. These services have to be adequate as to the excellence of the patient care, as to size of the training facility, and as to the equipment available.

It is also essential that an adequate number of patients with a large variety of conditions are treated by these services in order to provide for the completeness of the teaching program.

The following services are considered minimal in connection with the training of students in rehabilitation. A clinical inpatient and outpatient service in physical medicine and rehabilitation under the direction of a physiatrist should be established. More so, a reasonable number of beds should be allocated as an independent service for the treatment of inpatients. Physical therapy and occupational therapy service, and speech therapy should be established under the direct guidance and supervision of the physiatrist. Psychologic evaluation and guidance to the patient as well as to the clinical staff should be provided. Vocational counseling and social service given by appropriately trained personnel should be available. It is also necessary to provide for prevocational exploration and evaluation. These services should be on a full time basis as an integral part of the total rehabilitation program. Finally, it is highly desirable to have available prosthetic and orthetic services. If children spend extended periods in the clinical program, special educational facilities and teachers, from nursery school through high school, should be provided.

Teaching Facilities

The necessary facilities and equipment for the services already named should be available. In addition, an adequate research laboratory which can be used for teaching purposes is essential to fulfill the scope of the program.

Administrative Organization

The complexity of such a rehabilitation training program in a medical school makes it imperative to provide for an administrative organization which allows the proper coordination and integration of the teaching program and the patient services. This administrative organization must be structured to insure efficient and economical function.

It is recognized that some of the details of the plan of organization of such a curriculum have to be modified according to the needs of the corresponding school of medicine. It is also realized that certain minimal requirements have to be worked out to make the preceding proposal workable. These are:

A department of physical medicine and rehabilitation should be established and developed to the highest level of performance so that it can render adequate and efficient service to patients appropriate for teaching purposes. The department of physical medicine and rehabilitation in the medical school and in the teaching hospital should have independent status, or should have at least full administrative autonomy with a separate budget in order to serve its

purposes most effectively. The curricula as well as the services in physical therapy, and occupational therapy should be established as an integral part of this department, with appropriate provision for guidance from other medical specialties concerned with particular aspects of the paramedical health sciences. The other paramedical faculty and services should be administratively as closely associated as possible with the academic as well as with the service portion of the department.

Summary

It is believed that the proposed teaching program will be most effective if a competent physiatrist to direct the program, an adequate and competent staff, and adequate facilities for demonstrating the clinical management of patients with rehabilitation problems, are available. In addition, facilities for carrying on research should be provided in order to train medical students to understand and practice modern medical rehabilitation. Medical students are attracted to effective functioning medical programs and are discouraged and actually repelled from programs which are incomplete, inadequate, or inefficient. Because of the exceedingly strong support for most other medical programs, from many other agencies both voluntary and governmental, physical medicine and rehabilitation cannot compete successfully for the attention of the medical student nor attract young physicians into residency training unless it can offer comparable educational and research opportunities. This is demonstrated by the fact that presently only a low percentage of residencies is filled in the specialty of physical medicine and rehabilitation. Although there are many excellent opportunities for physiatric practice available in teaching institutions or rehabilitation centers or private practice, physical medicine and rehabilitation is not attracting a sufficient number of young medical students to enter this field. Brief or cursory exposure to rehabilitation is not attractive because it does not carry with it the understanding of the needs or potentialities of rehabilitation.

An inadequate program under the direction of a physician who is not competently trained in physical medicine and rehabilitation is more likely to deter than to encourage medical students to enter physical medicine and rehabilitation. Such a student does not even have the opportunity to observe the potential of rehabilitation when the teaching program is inadequate. It is only by the demonstration of the efficacy of a total rehabilitation program that the mature medical student recognizes the potential help to be derived by these methods.

In general, the practice of physical medicine and rehabilitation requires a physician of greater than normal maturity. The goals of rehabilitation are long-term goals achieved through prolonged therapy and management of the patient. The short-term goals and immediate and dramatic satisfaction, such as obtained by surgery, do not exist. Consequently the attraction in physical medicine and rehabilitation is for the medical student with a considerable degree of maturity, a higher than average social interest in the welfare of his patients, and a degree of dedication which will lead him to spend a considerable amount of energy in order to achieve the restoration of his patients. It appears that only through the completely developed teaching program herein described will medical students be attracted into this field. Therefore, it appears logical that the more extensive support of a few complete rehabilitation educational programs will in the long run be much more effective than the development of many small and limited programs. The number of schools thus supported will probably be limited by the availability of funds but the excellence of the teaching program should be the major criterion for determining eligibility for support.

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editorial

The Fovea Centralis of Rehabilitation Education

Elsewhere in this issue appears a sober appraisal of the teaching of rehabilitation in one medical school. Those readers who are interested in education technics and programs will immediately recognize in this article many excellent elements which with good reason the author is satisfied. However, because there are other programs which vary considerably from that described by Dr. Neu which also purport to teach rehabilitation, because of the inherent dangers in awarding the teaching of a subject to a department on the basis of its traditional claim to a large segment of the students' time, and because there are those who believe that the physician specialist in rehabilitation is not necessarily every patient's physician, additional comment on the subject of rehabilitation education should be made. These comments are in no sense to be construed as a "refutation" of Dr. Neu's thesis, some parts of which I believe to be most excellent and timely, but as expressions of an individual also interested in the teaching of rehabilitation.

Perhaps one reason for the divergencies of rehabilitation teaching programs is that the word, rehabilitation, means different things to different individuals. In order to define such a term in a fashion acceptable to all, vagueness has necessarily been resorted to in the construction of the definition. Most definitions of rehabilitation, it seems to me, share the fundamental inadequacy of that noted in Dr. Neu's paper. The dermatologist successfully struggling with a stubborn case of eczema, the ophthalmologist in extracting a cataract, the proctologist in snipping off an offending hemorrhoid all may survey this definition and aver solemnly that they also are engaged in the rehabilitation of their patients. Is this really the intent of the meaning of rehabilitation? If it is, obviously it should be no source of surprise to note that our confreres adopt the finger-tapping-the-skull gesture when we profess that, by our own definition, the special something we do is nothing really more than the routine day by day practice of medicine which every physician does. Surely the time has come that we no longer need couch this term of rehabilitation in generalities. It is either something of substance or it is nothing but shadow. If it is the latter, then it should be taught by the Department of Osmosis which exists in every medical school for the purpose of inculcating bedside manner, facility of medical expression and that which is commonly referred to as "good medical practice" in the mind of the medical students.

In actuality, most medical educators agree that rehabilitation should be taught in a definite, specific manner, the details of which may vary locally. The things for which

medical rehabilitation stand for are, for the most part, of recent interest in medical education. By virtue of this neogenesis, there necessarily will be confusion and divergence of opinions initially as to whom should be delegated the teaching of this topic. It is believed that few will disagree with the basic concept that any area of specialization in medicine requires a formal period of training for its practice. The same stipulation applies to medical rehabilitation as a specialty. Interest and enthusiasm alone, without training, are both inadequate and dangerous as a basis for either teaching or for clinical competence. The only area of medical training which thus far commits itself to the primary interests of medical rehabilitation is that of Physical Medicine and Rehabilitation. In some schools, however, there is no physiatrist on the faculty nor is there effort to obtain one, or the physiatrist is not consulted in the curriculum planning of rehabilitation teaching, or he is on the fringes of such planning. This seemingly indicates an unwillingness on the part of those responsible for such teaching programs either to recognize the training of the physiatrist or to reorient an admittedly complex scheduling of medical training to include his contributions. If the former be true, then the value of all other specialty training would seem to rest on the shaky foundation of traditional acceptance. If it is the latter, in the light of these changing times, one may legitimately wonder as to the validity of the threadbare excuse that there is no time available for such teaching or to have the teaching of medical rehabilitation awarded to a department on the grounds that it has time available to do so. The teaching of obstetrics is not done better by the orthopedist, nor the teaching of neurology by the ophthalmologist, nor the teaching of medical rehabilitation by the internist. Unless there be common understanding as to the confines of medical rehabilitation, it would seem that this important concept of treatment will founder if not be lost entirely in the uncertainties of what direction is forward.

The wonderful organ of sight is most sensitive to vision at the fovea centralis. Scarcely 3 mm. to the medial side is the optic disc which represents the confluence of all retinal nerves; this area is quite insensitive to light and is termed the blind spot. This would seem to be a propitious time to be certain that there is no blind spot in the teaching of medical rehabilitation.

— Donald L. Rose, M.D.

survey of selected literature

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ACTA ORTHOPAEDICA SCANDINAVICA. *Vol. 30 (Part 2), 1960.*

◇ **A New Method for Assessment of the Static and Dynamic Weight Bearing of the Foot. H. Wetzenstein. pp. 91-100.**

In this report a new method for the quantitative and qualitative measurement of the weight bearing of the foot in the shoe is described. The measuring apparatus is made of a stiff spring balance, consisting of a plate which is carried by three cantilevers. On each cantilever are mounted two strain gauges, one on the upper side and one on the lower side. The cantilevers have a certain flexibility, and it is this deflection during loading by force which influences the strain gauges. The resistance-changing produced in them are directly recorded by means of mirror galvanometers. By means of the three curves which are obtained, the total vertical load is calculated, and also the point of action of the load. The resolution of the weight-bearing load which corresponds to the point of action of the load on the weight-bearing surface migrates over this surface in a characteristic way during the weight-bearing face of a step. Whether this migration is sufficiently significant to allow a demarcation of different weight-bearing types will be investigated during the continuation of the work. It is also possible to make similar measurements even under the forepart of the foot's weight-bearing surface.

◇ **Uptake of S^{35} in the Intervertebral Discs after Injection of S^{35} Sulphate. An Autoradiographic Study. H. J. Hansen, and S. Ullberg. pp. 84-89.**

This study was undertaken in order to localize radiosulphur in the intervertebral discs and to follow the changing autoradiographical distribution patterns over a period of time. Five piglets were injected intraperitoneally with a dose of 1 mC carrier-free S^{35}-sulphate per kg. bodyweight. After injection they were killed at intervals of 1.5 hours, 6 hours, 24 hours, 4 days and 16 days. Immediately after the animals were killed specimens were made from the lumbar vertebral column. It was found that one and a half hours after the injection of S^{35} radioactivity is apparent throughout the entire specimen. Six hours after the injection of S^{35} activity is more distinctly concentrated to the epiphyseal cartilage, the inner portion of the annulus, and the periphery of the nucleus. One day after injection the appearance has changed radically with nearly complete disappearance of activity from the diaphysis of the vertebral body and concentration in the three sites already mentioned. The most important chemical property of the nucleus is its content of polysaccharides. Several authors have shown that both chondroitin sulphate and keratosulphate are present, and that S^{35} labelled sulphate is taken up by cartilage mainly as ester sulphate in chondroitis sulphuric acid. Radiosulphur can also be recovered from keratosulphate. The manner in which S^{35} was incorporated in the nucleus pulposus in this experiment indicates that synthesis of the two sulphomucopolysaccharides of interest takes place in the peripheral zone. The incorporation of radiosulphur illustrates the metabolically most active components in the intervertebral space — the periphery of the nucleus, the inner portion of the annulus, and the epiphysis of the vertebral body.

On the prognosis of rheumatoid arthritis. *Jonsson, E. p. 115.*

Correction of spastic equinovarus deformity in elderly hemiplegics by tenotomies. *Fried, A. p. 149.*

ACTA PHYSIOLOGICA SCANDINAVICA. *Vol. 50 (Part 3-4), 1960.*

◇ **Vasomotor Reflexes from Muscle. C. R. Skoglund. pp. 311-327.**

Casual observations of blood pressure changes during muscle stretch drew attention to the possible role of muscle proprioceptors in vasomotor control. An attempt has been made to

throw some light on this problem by studying the effects of mechanical muscle stimulation, and also of electrical excitation of muscle afferents, on systemic arterial pressure and on regional hindlimb circulation, in anesthetized limbs of decerebrated cats. Typical changes in systemic arterial pressure — viz., an initial depressor effect, followed or not by a more or less pronounced pressor effect — could be elicited by moderate stretch of different fore- and hindlimb muscles. The effects, which were dependent on intact nerve connections, appeared at tendon loadings of 100-300 gm. and could also be produced by slight pressure on the muscle belly or tendon. The conclusion that these effects were reflex responses mediated from mechanoreceptors in muscle was further substantiated by comparative experiments which, i.e., showed the divergent responses to various types of painful stimulation applied to the muscle. For eliciting systemic depressor effects by electrical stimulation of muscle afferents, stimulus strengths of more than 10 times the threshold of group I fibers were required; this implies that no depressor response was observed until after maximal activation of group I and II fibers, group III fibers were being excited. By simultaneous recording of the local arterial pressure in the lower hindleg, perfused at constant rate, the occurrence of regional vasomotor effects — predominant vasodilatation or constriction — could be demonstrated during electrical or adequate activation of muscle afferents. Studies of blood flow in muscles by inserted thermocouples were also performed.

◇ **Intermittent and Continuous Running. (A further contribution to the physiology of intermittent work). Erik Hohwu Christensen; Rune Hedman, and Bengt Saltin. pp. 269-286.**

Intermittent running on a treadmill at a speed of 20 km./h. (12, 4 miles/h.) is analyzed and a comparison between this work and continuous running at the same speed has been done. The present results are in agreement with the assumption that stored oxygen plays an important role for the oxygen supply during short spells of heavy work. When running intermittent 6.67 km. in 30 min. (effective work 20 min. and rest 10 min.), a trained subject attained a total 0_2 uptake of 150 l. With an 0_2 uptake of 0.4 l/min. at rest standing at the treadmill, or 4 l per 10 min. of rest, 146 l 0_2 are due to the 20 min. of work. The actual uptake at work was only 101 l and if normal values are assumed during rest pauses, a deficit in oxygen transport of 45 l arises during the 20 min. of actual work. This quantity will be taken up during the 120 rest pauses of 5 sec. each. Two-thirds of the oxygen demand during the 120 work periods of 10 sec. each will accordingly be supplied by oxygen transported with the blood during work, and one third will be covered by a reduction in the available oxygen stores in the muscles, which in turn will be reloaded during the subsequent 5 sec. rest periods. Respiratory and circulatory functions at intermittent and continuous running with special reference to maximal values are discussed. Research on intermittent work may open up a new field in work physiology.

The activity of muscle receptors in the kitten. *Skoglund, S. p. 203.*

Central connections and functions of muscle nerves in the kitten. *Skoglund, S. p. 222.*

The reactions to tetanic stimulation of the two-neuron arc in the kitten. *Skoglund, S. p. 238.*

Circulatory and respiratory adaptation to severe muscular work. *Åstrand, I.; P. O. Åstrand; E. H. Christensen, and R. Hedman. p. 254.*

AMERICAN JOURNAL OF PHYSICAL MEDICINE. *Vol. 39, Oct. 1960.*

◇ **Comparative Effects of Insulated Boots on the Circulation in the Lower Extremities. N. C. Birkhead; K. G. Wakim, and E. V. Allen. pp. 184-189.**

The effectiveness of two types of insulated boots was compared with respect to their ability to conserve body heat in the lower extremity. Using a plastic boot and a thermo-sock type of boot, the authors hoped to find a safe convenient method of increasing blood flow to the ischemic extremity. Of the patients with peripheral vascular disease, 75 per cent showed an increase in skin temperature of the toe of 1 degree centigrade or more during application of the plastic boot, while 31 per cent of the same group showed an increase of toe temperature of 1 C. or more during application of the thermo-sock. In 10 per cent of the 16 patients with peripheral vascular disease, there was an increase of blood flow of four per cent to 129 per cent of control values with use of the plastic boot, while with the thermo-sock there was an increased flow in only five, ranging from three to 20 per cent of control values. The response to use of either the plastic boot or thermo-sock was not statistically significant, nor was it

constant or predictable. The authors conclude that the use of an insulating boot has limited value in the treatment of arterial insufficiency of the lower extremity.

◇ **The Relation of Strength and of Temperature to Contraction of Skeletal Muscle. S. H. Walker. pp. 191-215.**

This paper is the first of two papers on muscle contraction. It discusses the structural basis of muscular contraction, the energy changes accompanying shortening and stretch, the electrolyte shifts occurring in the twitch response and the active state of skeletal muscle. Present theory holds that the active contractile component of muscle fibers are alternating filaments of actin and myosin. During contraction cross bridges form between the actin and myosin with the actin filaments being drawn in between the myosin filaments. When muscles shorten, work is performed and heat is produced in excess of the heat produced in isometric contraction. The production of excess heat shows that shortening muscle does not behave as an elastic body, yielding potential energy. When contracting muscle has work done upon it in the form of stretch, heat production is reduced. It is suggested that in the development of tension in the twitch response, there is first a rapid formation of cross bridges, in the latency relaxation period. During the contraction process, in the next phase, new reaction sites are made available for the cross bridges, resulting in a slow step-wise sliding of actin filaments between myosin filaments. In the active state of muscle, the full potential of contractile force is present. The author proposes that the full potential of the active state is not reached in the normal twitch. Compliance is reduced during twitch causing a tremendous resistance to stretch so that with stretch applied to the contracting muscle tension develops which exceeds the normal contractile force of the muscle.

Neurophysiological principles in the rehabilitation of physically disabled persons. *Missiuro, W. p. 171.*

Methods of recording movement. *Hellebrandt, F. A.; E. T. Hellebrandt, and C. H. White. p. 178.*

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AMERICAN JOURNAL OF PHYSCIAL MEDICINE. *Vol. 39, Dec. 1960.*

◇ **Influence of Cycloid Vibration Massage on Trunk Flexion. W. Bierman. pp. 219-224.**

Cycloid massage was administered to the paravertebral area of the back and the posterior aspects of the leg continuously for one-half hour in 152 individuals. The muscle-relaxing effect of the massage on the trunk and leg extensors was determined by measuring the fingertip to floor distance during maximum trunk and hip flexion with knees extended before and after massage. The treated group was able to bring their fingertips 2.8 inches closer to the floor following massage, while in the control group there was an increase of 0.28 inch.

◇ **Comparative Work Stress for Above-Knee Amputees Using Artificial Legs or Crutches. W. J. Erdman, II; T. H. Hettinger, and F. Saez. pp. 225-232.**

Energy consumption and pulse rate were recorded in nine above-knee amputees when walking with crutches or with an artificial leg. The walking speed with crutches did not differ from that with an artificial leg. In five of the nine persons energy consumption was less when walking with an artificial leg. One subject showed a greater energy consumption with a prosthesis. In the remainder the difference was not significant. In the subjects with an excellent gait the energy consumption during crutch walking was significantly higher while the reverse was true in the poor walkers. In all subjects the pulse rate during crutch walking was higher and the average difference between crutch walking and walking with a prosthesis was 38.7% ± 9.1%. After three to four minutes of using an artificial leg the pulse reached a plateau while with crutch walking the pulse rate continued to climb, indicating approaching fatigue. The conclusion is drawn that walking with crutches places a heavier load on the heart than walking with an artificial leg. Prescription of prostheses would, therefore, seem indicated not only for healthy amputees, but also for amputees with a heart condition.

The relation of stretch and of temperature to contraction of skeletal muscle: Part 2. *Walker, S. M. p. 234.*

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BRAIN. *Vol. 83 (Part 4), 1960.*

◇ **Tactile Localization. C. R. E. Halnan, and G. H. Wright. pp. 677-700.**

The authors tested visual and proprioceptive factors in tactile localization in 48 healthy young adults. The subjects were untrained, there were no practice runs, and each position was touched only once, using a bristle. One group was tested without seeing the extremity in question by indicating the point stimulated on a chart compared with pointing to the position on his own digit. A second group indicated the position verbally, and a third group was allowed to move the digit stimulated. It is apparent that visual and proprioceptive as well as tactile sensations are necessary for localization, and that learning or use is a factor. The detailed observations recorded here will be of definite value to physicians and therapists engaged in rehabilitation of all types of physical disabilities.

Internal carotid artery occlusion in young adults. *Humphrey, J. G., and T. H. Newton. p. 565.*

Congenital ocular motor apraxia. *Altrocchi, P. H., and H. Menkes. p. 579.*

The morbid anatomy of cervical spondylosis and myelopathy. *Wilkinson, N. p. 589.*

The ultrastructure of human myasthenic and non-motor end plates. *Bickerstaff, E. R.; J. V. Evans, and A. L. Wolf. p. 638.*

The individuality of the motor cortex. *Bates, J. A. V. p. 654.*

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BRITISH MEDICAL JOURNAL. *Vol. 2, Dec. 1960.*

◇ **On the Natural History of Falls in Old Age. J. H. Sheldon. pp. 1685-1690.**

The author has investigated the mechanism at play in the frequently observed liability of old people to tumble and often to injure themselves. This paper presents the results of an inquiry into 500 falls which happened to 202 individuals ranging from 50 to 85 years of age. The inquiry was directed at old people living at home, since the hospital population of old age has a heavy pathological bias, and, in addition, faces postural risks different from those of the community at large. There were 171 accidental falls (34%) in 25 individuals and are of importance in that they offer the main target for prevention. Stairs accounted for one third of the accidental falls, and the complete contrast between this fact and the small amount of time actually spent at risk is a measure of the great hazard that stairs present to old people. Next in importance came inadequate illumination. One hundred and twenty-five of the falls occurring in 58 individuals were classified as drop-attacks. In a typical attack the individuals suddenly and without warning fall to the ground, without loss of consciousness. So unexpected and so sudden is the incident that there is no time to prevent or to break the fall. Trips were responsible for 53 falls in 41 individuals, and they seemed to be directly associated with age for half of the subjects were in the 75-84 decade. Vertigo accounted for 37 falls in 26 individuals, a surprisingly small number, considering the fact that popular belief ascribes all unexplained falls in old people to an attack of giddiness. Falls due to disease of the central nervous system amounted to 27 falls in 21 individuals. Twenty falls in 17 individuals were the direct consequence of looking upward with the head thrown back. Eighteen of the 500 falls were the direct result of postural hypotension, 16 were due to mechanical weakness in one leg, and finally 23 falls remained unexplained. Though mainly a clinical description, the author advances some speculations concerning the fundamental relation between advancing years and increasing liability to fall. On the whole, the evidence suggests that the different modes of fall are varying manifestation of a more fundamental defect in the control of posture and gait. The weight of evidence strongly suggests that this defect is central and that possible peripheral factors, such as muscular wasting or sensory defect, are unlikely to play more than an adjuvant role, so that the structural basis of the clinical picture is to be sought in the areas concerned in the central control of gait and posture. It is well known that aging is accompanied by a loss of nerve cells from the brain. It is suggested that the fundamental factor underlying the senile liability to fall is a decrease in the number of healthy nerve cells available for the control of posture and gait.

Plastic-bag asphyxia in adults. *Johnstone, J. M.; A. C. Hunt; and E. M. Ward. p. 1714.*

Alleviation of intractable pain. *Gavin, R. p. 1917.*

Anabolic hormones in dermatomyositis. *Armstrong, A., and W. R. Murdoch. p. 1929.*

Schistosomiasis of the spinal cord. *Hutton, P. W., and J. T. Holland. p. 1931.*

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ERGONOMICS. *Vol. 3, July 1960.*

◇ **The Measurement of Sensory-Motor Performance: Survey and Reappraisal of Twelve Years' Progress. A. T. Welford. pp. 189-230.**

Perceptual and central organizing ability in sensory-motor performance can be appraised in five general areas. Central mechanisms for dealing with incoming signals behave as if there were a single channel handling one signal or a set of coordinated signals at a time. Central mechanisms do not act as a single whole, but as a chain with at least three links. Stimuli impinge upon the sense organs converting them into patterns of nerve impulses which are relayed to a *perceptual* mechanism in which integration and identification take place. Next, there is a *translation* mechanism concerned with the choice of action in relation to what is perceived. Finally, the *effector* mechanism such as hands and feet receives the previously coordinated and integrated signals to carry out the command. Speed and accuracy of making choices may be the result of the subject making a series of sub decisions each taking the same time until finally this translation process is completed and the effector organ receives the stimulus. Another supposition is that in the translation process of a signal, each half is examined for a clue to the answer and by a series of continuing halving of the most correct sides of the impulse, a decision is reached. A third proposal is that the subject compares each possible identification with the incoming signal with the decision to act taken when all identifications are made. Once the decision is made the control of movement by the effector organ must be considered. A reasonable explanation is that the subject considers all movements short of the far edge of the target and rejects all movements short of the near edge. The rejecting process gets finer and finer until the subject is on the target. Another variable in the speed and accuracy is discrimination of signals if the degree of choice is held constant.

The present study suggests a new perspective in job analysis. Action takes place in a matrix of decisions about signals rather than movements and it appears that central mechanisms function with less individual variation than do the peripheral. If the limiting factor in the speed and accuracy of laborers, white collar workers or executives is the load of decisions he must make before acting then in jobs not amenable to analysis by work study methods the analysis might better be done by increasing the "load" of decisions until performance is impaired.

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JOURNAL OF APPLIED PHYSIOLOGY. *Vol. 15, Nov. 1960.*

◇**Effect of Nutrient Supplements During Work on Performance Capacity in Dogs. D. R. Young; N. S. Shafer, and R. Price. pp. 1022-1026.**

Dogs were provided with either water, vitamins, phospholipids, or a complete nutrient mixture during treadmill running and their maximum aerobic work performance was measured. Work capacity improved with increased water intake; however, water taken in excess of two liters did not further increase work capability. Provision of water during work probably increased performance by improving temperature regulation. Milk, vitamins, and phospholipids were deleterious to performance. Glucose and protein were neither beneficial nor detrimental. The lactosemia and galactosemia associated with milk consumption during work suggests failure in enzymatic conversion of these compounds to more utilizable sugars.

◇ **Blood-Borne Vasodilating Agent from Ischemic Tissues. B. R. Freeburg, and C. Hyman. pp. 1041-1045.**

Plethysmographic measurements of forearm blood flow show a drop to minimal values immediately after release of arterial occlusion of a leg. This is soon followed by a somewhat greater and more sustained increase to values above the control. Prepacking the leg with blood before arterial occlusion, or exercise before occlusion, or a combination of these procedures does not significantly alter the response. Calculated peripheral resistance in the forearm follows the pattern implied by the blood flow changes. It is suggested that the initial vasoconstriction is a reflex whereas the later vasodilatation is due to a stable, circulating agent from the previously ischemic leg.

Measurement of human capacity for aerobic muscular work. *Billings, C. E., Jr.; J. F. Tomashefski; E. T. Carter, and W. F. Ashe. p. 1001.*

Physiological comparison of three types of ergometry. *Bobbert, A. C. p. 1007.*

Energy expenditure in level and grade walking. *Bobbert, A. C. p. 1015.*

Effect of training on eosinophil response of exercised rats. *Keeney, C. E. p. 1046.*

An improved procedure for determination of cardiac output by a conductivity method. *Hershgold, E. J.; S. H. Steiner and L. A. Sapirstein. p. 1062.*

Comparison of energy expenditure during treadmill walking and floor walking. *Ralston, H. J. p. 1156.*

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JOURNAL OF BONE AND JOINT SURGERY. *Vol. 42-A, Dec. 1960.*

◇ **Further Studies on Experimental Spondylitis and Intercorporeal Fusion of the Spine. C. R. Sullivan, and F. E. McCaslin, Jr. pp. 1339-1348.**

Interbody fusion by the surgical introduction of an inflammatory agent into a lumbar interspace was attempted in 74 adult mongrel dogs. Chemical irritants, proteolytic enzymes, and killed and living pyogenic bacteria were used. Solid intercorporeal fusion was obtained in all the 23 animals treated with living Staphylococcus aureus. Only one fusion was obtained in the 51 animals treated in other ways, and in this instance there appeared to have been a surgical infection in the interspace. A single inflammatory stimulus does not appear to be sufficient to induce interbody fusion in the dog's spinal column. A sustained process in which the intervertebral disc and cartilaginous plates are digested by proteolytic enzymes appears to be required to provide the chemical environment in which the proliferation of new bone and fusion can occur.

◇ **Bone Growth after Spine Fusion. J. T. H. Johnson, and W. O. Southwick. pp. 1396-1412.**

Using a rigid set of criteria which eliminated all but six cases from two hospitals, the authors measured bone growth after spine fusion. When spine fusion is unquestionably solid and fairly massive little increase in length occurs in the fused area. The microscopic and transient pseudarthroses are considered the most likely mechanism by which real increase in length or true angulation occurs. The studies of others which show an increase in length of practical value because they show what will happen to the average patient after spine fusion in early childhood; however, this type of data erroneously gives the impression that considerable growth occurs in the solidly fused spine. It was concluded that growth of a fused segment of the spine can occur only at the ends of the segment or at the site of gross or microscopic defects in the fusion plate.

Congenital malformation of the atlanto-axial joint with dislocation. *Brannon, E. W. p. 1377.*

The problem of the primary curve. *Cobb, J. R. p. 1413.*

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JOURNAL OF PEDIATRICS. *Vol. 58, Feb. 1961.*

◇ **Postinjection Sciatic Nerve Palsies in Infants and Children. F. H. Gilles, and J. H. French. pp. 195-204.**

The authors have reviewed 21 cases of sciatic palsy in pediatric patients which were associated with intragluteal injections. In all five cases with deep granulomas, the neurologic defect was functionally disabling. Six patients recovered completely in periods ranging from 24 hours to 13 months. In the remaining groups, four showed no recovery, and eight showed varying degrees of recovery. They believe neurolysis is not indicated early except in cases with palpable deep granuloma along the course of the nerve. It is recommended that injections be given at the lateral compartment at the junction of the distal third with the proximal two thirds of the thigh to prevent this complication in infants and children.

◇ **Viral Meningoencephalitis. J. N. Middelkamp; J. C. Herweg; H. K. Thornton; J. H. Brown, and C. A. Reed. pp. 205-210.**

Thirty-one out of 44 patients admitted to St. Louis Children's Hospital in 1958 who were presumed to have viral meningoencephalitis were studied in an attempt to establish an exact etiological diagnosis. In 26 cases (84%) the etiologic agents were identified, and in all but two virus was recovered from throat swab, stool, or spinal fluid. Two patients demonstrated diagnostic changes in complement-fixing antibody to mumps but no virus was recovered. Sixteen patients had poliovirus type I, 15 having paralytic disease. One poliomyelitis patient with no history of previous polio immunization died, one with no history of previous immunization had non-paralytic poliomyelitis. Three other patients with histories of two or three Salk vaccine immunizations showed paralysis which did not disappear. Seven patients had Echo virus type 9 meningoencephalitis. None showed paralysis or muscle weakness at any time. One child with Coxsackie B3 had transient weakness without permanent paralysis.

Symposium on the chemoprophylaxis of infection. I. Introduction and general principles. *Petersdorf, R. G. p. 149.*

II. The use of antibiotics to prevent infections by specific pathogens. *Petersdorf, R. G., and T. E. Woodward. p. 153.*

III. Prophylaxis of poststreptococcal sequelae and bacterial endocarditis. *Feinstein, A. R.; R. G. Petersdorf, and A. A. Browder. p. 164.*

IV. Protection against infection in susceptible individuals. *Petersdorf, R. G.; A. A. Browder, and A. R. Feinstein. p. 174.*

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NEW ENGLAND JOURNAL OF MEDICINE. *Vol. 263, Dec. 29, 1960.*

◇ **Oral Poliomyelitis Vaccine, Lederle — Thirteen Years of Laboratory and Field Investigation: An Interim Review. V. J. Cabasso; E. L. Jungherr; A. W. Moyer; M. Roca-Garcia, and H. R. Cox. pp. 1321-1330.**

In this report, a brief historical account of the development of Lederle oral poliomyelitis vaccine is given, and recent large-scale field trials with the vaccine are described. Six hundred eighty-four thousand persons have been fed the monovalent vaccine and about 500,000 the trivalent vaccine. Several hundred cases of poliomyelitis would have been expected if a similar population had been exposed to virulent poliomyelitis virus, and yet there has not been one case of nervous disorder or other illness in vaccinated subjects or contacts that could be attributed to the vaccine. The present Lederle strains of poliovirus multiply quite freely in the intestinal tract of vaccinated persons, are shed for several weeks and yield titers up to $10^{5.5}$ TCD_{50} per gram stool, particularly Types 1 and 3. Type 2 virus yielded lower titers per stool and was excreted by fewer persons. Interfamiliar spread of virus occurred quite readily, while extrafamiliar spread of the virus did not occur very often. Vaccine viruses were generally not recovered from the throat after ingestion of encapsulated vaccine. However, when liquid vaccine was swallowed, one, two or all three types of poliovirus could be isolated from the pharynx in a large percentage of the cases. Seroconversion rates for monovalent feedings varied in most cases from 83-91 per cent for Type 1; 54-87 per cent for Type 2 and 80-96 per cent for Type 3. Rates for the trivalent vaccine were of the same order. The trivalent vaccine is recommended to the public health service. The immunologic response to it is adequate.

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NEW ENGLAND JOURNAL OF MEDICINE. *Vol. 263, Dec. 1, 1960.*

Poliomyelitis in children: Experience with 956 cases in the 1955 Massachusetts epidemic. *Auld, P. A. M.; S. V. Kevy, and R. C. Eley. p. 1093.*

Reflections on the pathologic physiology of atherosclerosis. *Felch, W. C., and T. B. Van Itallie. p. 1125.*

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NEW ENGLAND JOURNAL OF MEDICINE. *Vol. 263, Dec. 8, 1960.*

Reflections on the pathologic physiology of atherosclerosis. *Felch, W. C., and T. B. Van Itallie. p. 1179.*

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NEW ENGLAND JOURNAL OF MEDICINE. *Vol. 263, Dec. 15, 1960.*

Reflections on the pathologic physiology of atherosclerosis. *Felch, W. C., and T. B. Van Itallie. p. 1243.*

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NEW ENGLAND JOURNAL OF MEDICINE. *Vol. 263, Dec. 22, 1960.*

Absence of the odontoid process. *Mann, G. T.; H. R. Bates, Jr., and H. H. Karnitschnig. p. 1300.*

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The Survey of Selected Literature for May 1961 was prepared by

Else A. Tophoj, M.D. *Odense, Denmark*	*for*	Acta Orthopaedica Scandinavica Acta Physiologica Scandinavica New England Journal of Medicine
Herbert A. Schoening, M.D. *Minneapolis*	*for*	American Journal of Physical Medicine Ergonomics Journal of Applied Physiology Journal of Bone and Joint Surgery
Harold M. Sterling, M.D. *Brighton, Mass.*	*for*	Brain Journal of Pediatrics
Osvaldo Miglietta, M.D. *Astoria, N. Y.*	*for*	British Medical Journal

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book reviews

HANDBUCH DER ALLGEMEINEN PATHOLOGIE. By *F. Buchner, et al.* Cloth. Price, $4.50. Pp. 434, with illustrations. Springer-Verlag, Heidelberger Platz 3, Berlin, Germany, 1960.

The five sections of this book deal with general radiation biology (Fritz-Niggli), radiohistology and radio-histopathology (Zollinger), biology and pathology of ultraviolet, visible, and infrared radiation (Miescher), superficial and deep effects of electric currents on the human body (Schwarz), and the significance of weather, season, and climate as pathogenic factors (de Rudder). Each section presents a critical review of the subject with an abundance of the factual and the concrete and a minimum of the speculative and the theoretical. It is especially gratifying to note de Rudder's discriminating treatment of bioclimatics, a field in which unfortunately much uncritical work has been done. Striking photographs are included in the section on electrical injuries by Schwarz; they should be examined by students of engineering as well as of medicine. The sections concerned with the ionizing radiations are packed with material of fundamental interest and practical importance. The text is thoroughly documented with references to original publications. Both subject and author indexes are provided. The book is highly recommended not merely as a key to existing literature but as a direct source of useful information.

CLINICAL ORTHOPAEDICS, NO. 17: Clinical Physiology and Pathology of Bones. Edited by *Anthony F. De Palma.* Cloth. Price, $17.50. J. B. Lippincott Co., E. Washington Sq., Philadelphia 5, 1960.

Clinical Orthopaedics is a well-known, regularly published series of symposia. It is produced under the auspices of the Association of Bone and Joint Surgeons.

Volume 17 is prepared as a tribute to Dr. Franklin Chambers McLean on the occasion of his 72nd birthday. The authors have all been associated in some way with Dr. McLean. There is a chapter devoted to a brief review of his career and one contains his bibliography. Another chapter written by Dr. Marshall R. Urist, guest editor for this occasion, is concerned with "The McLean Campaigns for Full-Time Academic Medicine."

The articles cover various basic aspects of musculoskeletal disease. They speak for the ability of their authors as much as they convey honor, respect and gratitude to Dr. McLean. The quality of this effort reflects the stature of the man to whom the book is dedicated.

The message of this collection is that basic scientific research is an excellent means to improve medical practice. The practitioner who studies these articles will have a new regard for the knowledge that is needed to properly practice medicine in the world of today.

The physiatrist will learn from this volume that his specialty will do well to explore and develop the basic sciences as they relate to neuromuscular diseases to better serve the patient and the medical profession.

This book brings more than the usual credit to its editors. *(Charles D. Shields, M.D.)*

THE MANAGEMENT OF FRACTURES AND SOFT TISSUE INJURIES. By the *Committee on Trauma,* American College of Surgeons. Cloth. Price, $5.00. Pp. 372. W. B. Saunders Company, W. Washington Square, Philadelphia 5, 1960.

This book is the combination of two former books, (1) An Outline of the Treatment of Fractures and (2) Early Care of Acute Soft Tissue Injuries. The former represents the seventh edition of it, while the latter is the second edition. Each book has been kept as its own entity indicated as Part I and Part II. Because of the combined effort, overlapping has been kept to a minimum consistent with good management. Since the average practitioner sees more soft tissue injuries than fractures and since nearly all fractures are accompanied by soft tissue injury, it seems to this reviewer that Part II ought to precede Part I.

This book is well-written, well-edited and is outlined for clarity of thought and delineation of the steps in treatment, in the order in which they should be accomplished consistent with the best medical management, as we know it today. The numerous contributors have made the editorial task most difficult but it has been accomplished in a most lucid manner. All who have played a part in this production are to be congratulated and thanked for this cooperative effort.

The description of each injury is discussed from the standpoint of how it may have been

incurred, and the importance of knowing how in the management of the patient and the treatment. This is followed by a description of the injury and the steps in proceeding with its treatment. All sections emphasize the importance of a clear airway, shock and an evaluation of the injury to determine the need of transfusions, antibiotics and innoculations. The described methods of management are not those of first aid as might be administered by a passerby but those of medical office practice or hospital emergency room care.

The book is practical for the senior medical student who is trying to summarize all his traumatic surgical training into usable knowledge as preparation for his internship. It is an excellent book for the intern to use in reviewing his daily care of the patients seen by him in the admission center or emergency room. The surgical resident should have it handy at all times as a quick reference to help in the management of early soft tissue injury and fractures. For the general practitioner, who may not have specialists and hospital facilities immediately available, this is a handy guide to life-saving procedures. It also would be equally valuable to the large community general practitioner and to all physicians who may only occasionally see acute trauma.

The many contributors have not seen fit to entrust their patients to physiatrists although they mention from time to time the use of physical and occupational therapy. They all seem to recognize the need for rehabilitation but most of them state that it must be under the surgeon's supervision. Perhaps the majority of the authors have not had the opportunity of cooperative effort with a physiatrist. In spite of this apparent failure to recognize physiatrics as a competent specialty of medicine, the reviewer feels that he must recommend this book to be handy for any physician.

LES MANIPULATIONS VERTEBRALES. By *Dr. Robert Maigne*. Cloth. Price not given. Pp. 246, with 261 figures. Expansion Scientifique Francaise, 15 rue St. Benoit, Paris VI^{e}, France, 1960.

The table of contents of this book is in the back, where American and most European readers are accustomed to find an alphabetical subject-index. There is no index, and little is said about the qualifications of either the author of the book or the writer of the preface. The book is in four parts containing (1) a general discussion of manipulative procedure with historical remarks and essential definitions; (2) technics of manipulation applied to the vertebral column; (3) indications for such manipulation, especially as to headache, neuralgias, lumbago, and sciatica, and (4) two supplementary chapters by Lescure and Waghmacker, one discussing in general terms the question of visceral organic effects of vertebral manipulations, the other setting forth in detail a number of technics for reeducating the patient disabled by spinal disorders. An appendix describes in detail the courses of exercise given in five typical cases.

This book deserves to be widely and attentively read by physiatrists and orthopedists. The text is extremely well written, clear, and objective. Taken together with the abundance of magnificent photographs and diagrams it gives, among other things, a complete exposition of methods which many people think of as the secret property of chiropractors. In reality, those procedures that are safe and effective have long been taught by such experts as the late J. B. Mennell, to whom the author gives due credit on page 10. What has been lacking has been just such a book as this, in which a clear and attractive style would be combined with freedom from cultist overtones. There still is need of a book that would do for the appendicular parts of the skeleton what this book does for the axial parts. Every medical student, whether he reads French or not, should take the time at least to turn the pages of this book. It deserves to be translated into Interlingua.

NICHOLS' MANUAL OF HAND INJURIES. By *H. Minor Nichols*, M.D. Cloth. Price, $11.00. Pp. 400, with illustrations. Year Book Publishers, Inc., 200 E. Illinois St., Chicago 11, 1960.

The author of this volume has succeeded in presenting quite a concise treatise on the treatment of injuries and infections of the hand. Within its four hundred pages, this book includes all of the information that a practicing surgeon needs to have for the satisfactory care of an injured hand. The medical student and the physical therapist will also find this book a valuable reference work.

The initial chapter on the anatomy of the hand is well presented, and the accompanying atlas of seven plates demonstrates very adequately the surgical anatomy of the forearm and hand.

Throughout the book, Dr. Nichols has constantly stressed the importance of simplicity of treatment and the danger of over-treatment. For example, with an incomplete severance of an extensor tendon, he correctly advises the least done to repair the tendon, the better; this is especially true if one can demonstrate at the time of the injury that function is not impaired. Dr. Nichols also cautions the inexperienced surgeon against undertaking any extensive reconstructive measures.

The author admits that there is often more than one way to treat a specific injury with equally good results. He gives the reader the benefit of his experience with various methods. In the treatment of tenosynovitis producing trigger fingers, DeQuervain's disease, or the carpal tunnel syndrome, Dr. Nichols fails to mention the use of local injections of cortico-

steroids; such local treatment in these conditions will often obviate the necessity for surgery.

With the use of diagrams, roentgenograms and preoperative and postoperative photographs of illustrative cases, the author indeed makes this treatise "come alive" for the reader. The postoperative management of the injured hand is well outlined, and this includes the proper use of physical therapy. *(George Phalen, M.D.)*

CHRISTOPHER'S TEXTBOOK OF SURGERY. Edited by *Loyal Davis,* M.D. Seventh edition. Price, $17.00. Pp. 1551, with 1597 illustrations on 810 figures. W. B. Saunders Company, W. Washington Square, Philadelphia 5, 1960.

The seventh edition of Christopher's Textbook of Surgery, edited by Loyal Davis, Chairman of the Department of Surgery, Northwestern University Medical School is enthusiastically recommended to students, residents, teachers, and practitioners as a comprehensive, authoritative, and highly accurate volume covering all phases of surgery. The aim of the book as stated in the preface is "to place before the student an interestingly told story of the facts and principles which should form the basis for his education in surgery, so as to stimulate him to continue his self-education throughout his professional life." The 82 contributing authors, each a respected leader in his field, have achieved this aim admirably well. At the end of each chapter, reading references to recent and historical contributions in the particular field under discussion furnish the student a fertile field for further propagation.

The book places proper emphasis on the relationship of the basic sciences to surgery. The first 180 pages are devoted to the basic sciences and the fundamental aspects of patient care. This upholds the principle that the surgeon of today must really be an internist who also has been trained in the technical facility of performing operations.

A new section on surgical judgment has been added. The chapters on infection, abdominal wall and peritoneum, surgery of peptic ulcer, the urinary tract, male reproductive system, the foot and amputations have been completely rewritten by new authors. The most recent advances are noted in these chapters as they are in all of the other chapters.

This book is an improvement over previous editions from the technical standpoint. The illustrations and photographs are magnificently done. The pages are excellently printed on good paper with legible type. The table of contents and index are clear, concise and easy to use.

All in all, the book represents a storehouse of knowledge, presented from the accumulated wisdom of the 82 outstanding contributing authors. It should find a very useful place in the library of everyone interested in surgery. *(E. R. D., M.D.)*

LUMBAR DISCOGRAPHY AND LOW BACK PAIN. By *Donald D. Bauer,* M.D. Cloth. Price, $5.00. Pp. 89, with illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1960.

In his introduction the author states that the monograph is offered as a demonstration of increased scope of knowledge which discography provides in the study of low back pain.

The book has a curious arrangement of chapters. First is an introduction, then an atlas of discograms, next practical applications, then technic, then research value of discography, and finally interest of the legal profession.

In the body of the book the author, a radiologist, makes his obvious point — namely, that discography is a direct examination of the intervertebral disc, while the conventional oil myelogram is indirect. Be it noted that the discogram is also an indirect examination. We are not concerned with disease of the disc *per se,* but rather with whether it is impinging in some way so as to put traction on a dorsal root. He is quick to admit that the discogram requires one needle for each disc, while the myelogram needs only one needle for all discs; that the time required for the discogram is up to three times as long as for the myelogram (but still what the author considers in the safe zone); that headache and pain occur following the discogram; and finally that more skill and fluoroscopic experience are needed for the discogram than for the myelogram.

I am going to comment first at the level of language and then at the level of understanding of disease. The language, like almost all current medical publication, is undistinguished and often inexact. The author has fallen into the habit of using the term with a current vogue, "discogenetic disease." The actual etymologic implication of this word is that of a disease which in some mysterious way generates intervertebral discs.

The author's shortcomings at the level of understanding of a symptom such as pain and of disease are more serious. Like many of his brother radiologists he seems to have adopted an interpretation of medicine based on "seeing is believing." Perhaps this is why radiologists so often strike out in clinical-pathologic conferences, even in cases in which a maximum of help would be expected to have been afforded by the submitted films. There is a clear confusion in this author's mind between some variety of disease of the intervertebral disc and the existence of a pain syndrome in the low back. He leaves the strong implication that one should consider malingering when a careful study of an appropriate number of discs reveals no evidence of disc disease.

No patient should be subjected to either myelography or discography who does not have evidence by competent neurological examination of radicular syndrome. Without such information one could examine all the discs in the lumbar spine with no clear idea of localization beyond the statistical fact that disease of this organ occurs frequently in this part of the spinal column. The author does not say what he would do if he discovered evidence of disc disease in a person who had no back pain, or what he would do next if a patient (as frequently happens) continues to report pain after careful removal of an intervertebral disc which had shown disease. *(Sedgwick Mead, M.D.)*

OCCUPATIONAL THERAPY IN REHABILITATION. A Handbook for Occupational Therapists, students and others interested in this aspect of reablement. Edited by *E. M. MacDonald*. Cloth. Price, $8.50. Pp. 348, with illustrations. The Williams & Wilkins Co., Mt. Royal & Guilford Aves., Baltimore 2, 1960.

Miss MacDonald is Principal, Dorset House School of Occupational Therapy. The contributors are either members of her staff or graduates of the Dorset House School. The book is intended to portray the philosophy of occupational therapy, its aims, its scope, its general place in the medical field. Principles are emphasized rather than exact technics, since these are presumed to have been studied in other texts. It gives the broad concept which physicians can appreciate and which may be the means of introducing them to the value of the field of occupational therapy. It discusses the approach to the patient and the examination of the patient, basic principles of physical treatment in order to re-educate for function following trauma, neuromuscular involvement, or the arthritides. The importance of occupational therapy for psychiatric conditions is given good space. Problems of children occupy two parts of the book. The administration of a department of occupational therapy in its various locations, its relationship to physical therapy and to social service is well developed. It has a good bibliography.

Physicians, occupational therapists, students of occupational therapy, or anyone who is involved with the management of patients will find this book very enlightening. It is both a text and a reference book. I am pleased to recommend it. *(Frances Baker, M.D.)*

POSTURAL FITNESS: Significance and Variances. By *Charles LeRoy Lowman*, M.D., and *Carl Haven Young*, Ed.D. Cloth. Price, $7.50. Pp. 341, with illustrations. Lea & Febiger, Washington Sq., Philadelphia 6, 1960.

This is a book to be added to the limited but growing number of textbooks for students in adaptive physical education. It was written by a teacher of physical education together with a well-known orthopedist who has long been interested in physical education. A general discussion of the need for recognition of problems of posture precedes a more detailed description of various types of deviations of posture. These are well illustrated by descriptions and photographs. Exercises for specific conditions are described which, for the most part, are the standard ones for improving flexibility and strength. Some are useful in physical therapy, especially for correction of posture in certain cases. A strong plea is made for closer cooperation with physicians in dealing with posture problems. The last part of the book contains outlines of exercises for groups; these are graded for elementary school through high school. In the light of the increase in interest in exercise for the general population, it is important for physical educators to be more thoroughly grounded in all types of exercise. This text will be helpful for teaching problems of postural deviations to students in physical education. The appendix contains lesson plans and other aids for teaching. *(Donald J. Erickson, M.D.)*

EARLY IDENTIFICATION OF EMOTIONALLY HANDICAPPED CHILDREN IN SCHOOL. By *Dr. Eli Bower*. Cloth. Price, $5.50. Pp. 120. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1960.

As the title indicates, this is a report on a study of a process for screening children who are, or who may be developing emotional problems. The study has been carried out on over 40,000 children in California schools, over a six-year period, and is reported by Dr. Eli M. Bower, Deputy Director, Liaison and Prevention, California State Department of Mental Hygiene. The special teacher testing processes have been furnished by the California Board of Education.

Much of the process as developed can be effectively carried out by the teachers, so that they can make better estimates of the mental health of their individual students. Early detection of personality and behavior disorders can be demonstrated by these methods so that in many instances clinical help will thwart or clear up an extension of the disorders. These processes carried out by the teachers have been found to usually correlate with the finding on the same students when made with other technics by psychiatrists, clinical psychologists and psychiatric social workers. The best results were obtained with teachers who voluntarily took a course to learn more about the testing technics and aims of the study.

Dr. Bower urges that the terms "emotionally handicapped" replace either the terms, "emotionally disturbed" or "socially maladjusted." It is his opinion that the term

"handicapped" has a more lasting and persistent quality, while "disturbances" are often transitory or temporary. Furthermore, "emotionally disturbed" has a connotation of formal psychological or psychiatric appraisal which, in these instances, is not always wise.

Inability to learn, unsatisfactory inter-personal relationship, inappropriate behavior, unhappiness and repetitive illness are the significant characteristics of children — which indicates a need for closer scrutiny. Differentiation between differences and pathology, relationship of definition to personal theory, degree of emotional handicap and problems in early identification of emotionally handicapped children are discussed. Studies by other investigators and their results are reported upon. The methods of this study and research results for screening are developed. Appendices give the three detailed tests provided by the California Board of Education which the teachers use. There is a discussion as to how the teachers can use the results in screening and how this can be and is correlated with the work of a clinical team.

It would appear to this reviewer that the material presented should not be embarked upon by just any teacher who reads of the work and becomes interested. It would seem that the teachers who are to do the studies should have special indoctrination in the processes, and their work should not be done until a full program is developed, with a clinical team of psychologists and psychiatrists who not only understand the process, but are included in the team. They will need to carry out other tests in at least selected instances, such as Wechsler Intelligence Scale for Children, Stanford-Binet, etc., be available for advice, guidance or even therapy. As a matter either of caution or information it was pointed out that some teachers also sometimes have personality conflicts which they must understand before they can do unbiased evaluation of the tests they are giving to their students.

This book should have specific interest to all persons concerned with these important problems in the field of mental health. To other physicians or therapists, except for a general knowledge that such studies are progressing, the details would have little interest. *(Nila Kirkpatrick Covalt, M.D.)*

PRINCIPLES OF ORTHOPAEDIC SURGERY. By *Paul C. Colonna*, M.D. Revised edition. Cloth. Price, $22.00. Pp. 799, with illustrations. Little, Brown and Company, 34 Beacon St., Boston 6, 1960.

Dr. Colonna's eminence in his field is further enhanced by this textbook of Orthopedic Surgery. His years of experience in teaching and practice form the basis of a work which is directed toward the general practitioner as well as to the specialist. In keeping with the present trend of increasing frequency of accidents, greater emphasis has been placed on trauma in this revision of an earlier edition.

Following introductory chapters on physiology and pathology of bones and joints, each region of the body is considered separately. In general, the discussions include applied anatomy, diseases, soft tissue injuries, fractures and treatment. Reference is made to the use of physical therapy, but only in the general terms of "baking, massage and exercise." The importance of ancillary personnel is mentioned briefly.

A chapter titled "Principles of Apparatus" is disappointing in that the bulk of space is directed to a detailed discussion of methods of applying plaster and traction, plus a review of the conventional braces and supports. No mention is made of the new fields of functional bracing or prosthetics. Little emphasis is given to the present-day uses of steroids and muscle relaxants.

The format is excellent, the style sufficiently informal to make easy reading without sacrificing concise descriptions. A wealth of photographs, sketches and diagrams both supplement and complement the text. Bibliographies are comprehensive for the majority of chapters.

The volume will undoubtedly find its place with those basic texts which are in greatest demand. *(Harriet E. Gillette, M.D.)*

MAN'S POSTURE: ELECTROMYOGRAPHIC STUDIES. By *J. Joseph*, M.D. Cloth. Price, $5.50. Pp. 88, with illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1960.

This book is a well-thought-out, well-executed and controlled electromyographic study of fundamental principles of human posture. This treatise consists of only eight short chapters, but so concisely and clearly written that all pertinent details are given. There is a lengthy, almost eight-page, list of references, besides the indexing.

Dr. Joseph standardized his studies, using skin electrodes all of the same size, placing them in identical positions on every subject (male and female), and on every muscle tested. Every subject also stood in the identical "at ease" position. In reporting on his findings, he also discusses and compares them with the previous reports of other investigators. In many instances he has also duplicated their experiments for confirmation. There is generally much agreement. This agreement clearly refutes long-held views regarding muscle tone as it relates to posture — or, man's response to gravity. It has long been held that man's response to gravity in the fundamental principles of posture is maintained by a balance of muscle tone in a state of tension. Electromyographically then, those muscles thought to be in tone should have shown action potentials, but none of the investigations have found such potentials, and these muscles are

as silent as are muscles which are relaxed when an individual is lying down.

The first chapter discussed man's posture. Accepting either Webster's International Dictionary or the Shorter Oxford Dictionary for the definition of posture ". . . especially the position and carriage of the limbs and body as a whole," man's posture is discussed as to the general, the correct and the variations. The different relationship of body parts as to other bipeds is described and illustrated for comparison. Dr. Joseph states that man's posture is unique because of the totality of the relations between the different segments of the body, so that the posture of man can be defined in general terms. Basic to this study and its interpretation is to know the line of weight. This is in relation to the transverse areas of rotation of the joints of the vertebral column and the lower limbs. There is a fairly general agreement among all researchers as to the center of gravity.

Apparatus and standardization of the method and use of the electromyograph are discussed in Chapter Two. Relaxed muscles and the question of muscle tone is the important subject in Chapter Three. Dr. Joseph advances the opinion, "that the term *muscle tone* or *tonus* should be discarded, but if it is used, it should refer only to the response of a skeletal muscle to stretch." This conclusion has been reached as one of the results of this research. Theoretical Considerations, Investigation and Results, Other Investigations, Discussion and Conclusions Relating to the Leg Muscles are reported in Chapter Four. The same outline is followed in Chapter Five concerning the thigh muscles; the hip muscles in Chapter Six; the Posterior Vertebral Muscles in Chapter Seven. Chapter Eight consists of comments and conclusions.

Sufficient evidence is presented and confirmed to show that the common and long-held concept that posture is maintained by the contracture of many muscle groups is erroneous. Rather, the following conclusions were reached. The line of weight in almost all subjects falls in front of the ankle joints when standing "at ease." This position was proved to be maintained by contracture of the larger calf muscles, especially the soleus, the appropriate ligaments of the knee and hip joints, and some part of the posterior vertebral muscles. The hamstrings are contracted in some. Swaying at the ankle flexion of the knee or hip, or movements about various segments of the vertebrae do bring on activity, usually in the extensors as they resist the force of gravity, but these motions are not those which maintain posture. The term "postural muscles" therefore has been incorrectly used. These concepts are not only interesting, but undoubtedly have many practical applications. Studies are still to be done on the abdominal and anterior vertebral muscles, while the amount of tension that ligaments can stand before rupture also needs further investigation. *(Nila Kirkpatrick Covalt, M.D.)*

THE LIST METHOD OF PSYCHOTHERAPY. By *Elizabeth Sher, et al.* Cloth. Price, $7.50. Pp. 258. Philosophical Library, Inc., 15 E. 40th St., New York 16, 1960.

The title refers to the psychotherapeutic method of Jacob S. List and consists of six chapters, each having been written by a student of his and each dealing with a separate aspect of the List philosophy of treatment. The authors, E. Sher, A. Messing, E. Messing, T. Hirschhorn, E. Post, and A. Davis, are all former patients, former students and all have doctorates in education.

The orientation is truly liberal and informal in both theory and practice. There is particular emphasis on warmth and the therapeutic use of the self. Of note is the development of the reception room into a therapeutic community. Its informality and hominess reassures the new patient, but much more important is the encouragement of friendships among patients with the opportunity for viewing social behavior and the reinforcement of the work of individual therapy.

The basic principle of treatment is described as substituting a "program for a problem." There is an emphasis on activity and decision-making with questioning and discussion coming later. An unusual aspect of this is the emphasis on furthering one's education particularly formal college work if the ability for it is present.

The attitude throughout the book is of an intense, personal, and therapeutic approach to psychotherapy with good sophistication and self-awareness and little interest in abstraction or theoretical formulation. The approach is stimulating, clearly consistent with the now greatly broadening concept of psychotherapy and the book will be helpful, I am sure, to all who are in some way engaged in psychotherapy in its broadest sense. *(J. M., M.D.)*

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2. Manuscripts **must be** in the office of the American Congress of Physical Medicine and Rehabilitation, 30 N. Michigan Ave., Chicago 2, not later than March 2, 1962.

3. Contributions will be accepted from interns, residents, graduate students in the pre-clinical sciences, and graduate students in physical medicine and rehabilitation.

4. The essay must not have been published previously.

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7. The winner shall receive a cash award of $200.

8. The winner shall be determined by the Awards and Prizes Committee of the American Congress of Physical Medicine and Rehabilitation.

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